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HYPERTONIC DEHYDRATION AND SUMMER DIARRHOEA

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Diarrhoeal disorders are a major cause of morbidity and mortality in African children, especially during the summer months. Severe attacks occur mostly in children suffering from protein malnutrition.[1] Hypotonicity of the extracellular fluid occurs frequently in these dehydrated subjects.[2] Of the 1,000 children with diarrhoea and dehydration admitted to the paediatric wards of this hospital every year, 26·5% have serum-sodium levels of less than 125 mEq. per litre, while 60·5% have normal levels (126 – 144 mEq. per litre).

This leaves 13% who have serum-sodium levels of 145 mEq. per litre or more. These children are selected from a further 1,500 cases who receive intravenous therapy for dehydration in the out-patient department. Thus the actual incidence of hypertonic dehydration is probably 5 or 6% of the total number of infants and children attended for dehydration at this hospital. This form of dehydration has been reported with increasing frequency in recent years, particularly from the USA,[3-6] and its incidence, in dehydrated infants, ranges from 16 to 25%. In one series hypertonic dehydration was present in 17 (53%) of 32 children suffering from diarrhoea.[7] This high incidence was attributed to the oral administration of milk formulae high in solute content or to the use of oral aqueous solutions containing excessive amounts of electrolytes.[7] In this country Bowie *et al.*,[8] from Cape Town, reported an incidence of 34%.

In this paper we report 11 African children with hypertonic dehydration (5 of whom were briefly described previously[9]). It will be shown that neither excessive loss of water nor ingestion of concentrated saline solutions or milk formulae is likely to be the sole aetiological factor in the pathogenesis of this type of dehydration.

MATERIAL AND METHODS

The 11 children were selected, during a period of 3 months, from among 160 ward patients suffering from gastro-enteritis. A serum-sodium level of 145 mEq. per litre or more was the main criterion for inclusion in this series. This level was chosen in the light of two studies of normal serum-electrolyte levels carried out at this hospital. In one[10] the mean serum-sodium level was 136·3 (±4) mEq. per litre and in the other[11] 132·3 (±6) mEq. per litre. In the latter study the average serum-potassium level was 5·5 (±0·8) mEq. per litre, the average plasma-chloride level 98 (±7) mEq. per litre, and the average carbon-dioxide combining power 18·7 (±4) mEq. per litre. Blood-urea levels in normal controls were always less than 35 mg. per 100 ml.

The state of nutrition was assessed clinically. A child was considered to be malnourished if one or more of the following stigmata were found: (1) Patchy hypopigmentation or an acute nutritional dermatosis.[12] (2) Sparse straight hair with evidence of depigmentation. (3) Nutritional oedema. (4) A body weight below 40% of that expected for age; in the absence of normal weight standards for African children, we have compared the weights of our patients with those of normal American children.[13]

The degree of fluid loss was assessed by the difference between the patient's weight on admission and on discharge (the average stay in hospital was 9 days).

The following investigations were carried out on admission to hospital: Serum sodium and potassium were determined by means of an EEL flame photometer, plasma chlorides by the method of Schales and Schales,[14] carbon-dioxide combining power by the method of Van Slyke,[15] and blood urea by the urease method. Serum-salicylate levels were determined in 6 of the patients on admission. The urine was examined microscopically and for protein content and specific gravity, and in cases 9 and 10 the total urinary excretion of sodium, potassium and chloride was assessed over a period of 48 - 66 hours after commencement of therapy.

Treatment consisted of intravenous administration of 0·2% sodium chloride in 5% invert sugar (Na 34 mEq. per litre) in quantities of 80 - 100 ml. per lb. body-weight per 24 hours. Potassium chloride (27 mEq. per litre) was added to the intravenous infusion when the urinary output was adequate and provided that the serum-potassium level was not raised. Oral milk feeds were withheld until electrolyte levels returned to normal.

All patients received chloromycetin palmitate, 30 - 50 mg. per lb. body-weight per day, in divided doses. If there was an associated respiratory-tract infection, penicillin was administered in doses of 250,000 units by intramuscular injection every 6 hours.

CLINICAL AND BIOCHEMICAL FINDINGS

Some of the clinical and biochemical data of the 11 patients are summarized in Table I:

The age of the patients ranged from 2 to 10 months; 8 of them were males. The maximum rectal temperature exceeded 102°F (39°C) in 7 patients. In 7 patients the skin turgor was normal or only slightly impaired; in 3 there was a 'doughy' feel of the skin and subcutaneous tissues; in 1 (case 3) the skin was markedly inelastic. The state of nutrition was clinically good in 7 patients, fair in 2, and poor in 2, but none of the cases showed the features of kwashiorkor (protein malnutrition). Five patients weighed more than the average American child of the same age,[13] while only 2 were below the 3rd percentile.

In 9 of the 11 patients there were some neurological abnormalities such as irritability, stupor, tetany, coma or convulsions.

TABLE I. CLINICAL AND BIOCHEMICAL DETAILS OF 11 CHILDREN SUFFERING FROM HYPERTONIC DEHYDRATION

Case No.	Age (months) and Sex	Weight (1) On admission (2) On discharge (lb. and oz.) A	Degree of Dehydration B	Hyperpnoea C	Maximum Temperature °F	Skin Turgor D	Neurological Signs E	CSF Protein (mg. per 100 ml.) F	Respiratory System Involvement G	Clinical State of Nutrition H	Blood Electrolytes (mEq./litre) Na I	Cl J	K K	HCO_3 L	Blood Urea (mg./100 ml.) M	Serum Salicylate (mg./100 ml.) N	Haematocrit PCV% O	Haemoglobin (g./100 ml.) P	Urine S.G. Q	Urine Prot. R	Urine Casts S	Remarks
1	4 M	(1) 10, 12 (2) 11, 9	7	+	103	Sl. Imp.	Irr. HPC	—	Nil	G	156 132	142 100	4·8 3·6	6·7 16·2	74 13	—	34 35	12·0* 11·8†	1020	+ + +	Gran. (num.)	
2	10 F	(1) 22, 3 (2) 24, 1	8	+	106	Doughy	S. Coma. Hyp. Conv.	24	Nil	G	149 137 130	128 108 101	4·7 4·7 4·9	5·3 12·1 —	170 41 <10	—	41 31 34	13·5* 9·4† 10·7††	1008	+ +	Gran. (occ.)	
3	4 M	(1) 12, 14 (2) 14, 1	8	+	103	Poor	None	—	Creps. (*b*)	G	146 137	110 101	4·4 3·5	5·9 21·9	280 32	<5	32	10·4* †	—	+ +	Nil	Rickets
4	4 M	(1) 11, 14 (2) 10, 3‡	14‡	+	102·6	Normal	Irr. + MT Tet.	—	Br. pn. (*c*)	F	153 134 155	121 98 124	5·0 4·6 4·7	11·6 16·0 7·5	160 18 105	<5	34 35	* † 11·0‡ 11·0†	—	Nil	Gran.	Rickets Relapse Died (Fig. 1)
5	5 M	(1) 12, 7 (2) 13, 12	10	+	105	Normal	Conv.	31	Br. pn. (*d*)	F	149 134	114 106	5·0 5·4	9·5 21·7	106 14	—	36 33	11·7* 10·2†	1008	+	Nil	Rickets
6	7 M	(1) 16, 2 (2) 17, 8	8	+	105	Sl. Imp.	Conv.	58	Creps. (*e*)	G	162 144	148 111	5·5 5·4	6·5 18·4	290 <10	<5	27	9·7* †	—	+ + +	Nil	Salmonella enteritis
7	4 F	(1) 12, 9 (2) 14, 13½	15	+	101	Sl. Imp.	Stup.	—	Nil	G	145 137 134	118 99 101	5·4 3·7 5·7	— 26·6 19·2	280 18 10	<5	29 30 30	10·5* 10·6† 10·6††	—	Trace	Nil	
8	6 F	(1) 12, 3 (2) 12, 14	5	+	105	Normal	Irr. Stup.	24 (*a*)	Nil	P	146 137	118 102	4·2 3·5	6·0 18·2	152 31		40 31	13·7* 10·9†		Trace	Not exd.	Table II
9	4½ M	(1) 13, 4 (2) 14, 8	9	+	100	Doughy	S. Stup.	—	Creps. (*f*)	G	150 134	126 106	3·1 5·0	5·0 18·6	182 17	<5	35 26	12·5* 8·3†	1020	+ + +	Hy and Gran.	Fig. 2
10	5½ M	(1) — (2) 11, 14	—	+	101	Normal	None	—	Otitis Media	P	156 130	114 99	3·8 6·0	4·5 19·7	76 57	<5	32 34	11·4* 11·6†	1007	+	—	Salmonella enteritis Rickets
11	2 M	(1) 8, 5 (2) 9, 3	10	+	100·5	Doughy	Drows.	—	Nil	G	148 134	106 98	4·7 —	8·7 17·9	103 20	—		* †				

Column A: ‡ During a relapse.
Column B: Based on column A, [(2)—(1)] ÷ (2) expressed as percentage. ‡ During a relapse.
Column C: + = Present.
Column D: Doughy = Doughy feel. Sl. Imp. = Slightly impaired.
Column E: Irr. = Irritable. Conv. = Convulsions. Stup. = Stuporose. Hyp. = Hypotonic. Tet. = Tetany. Drows. = Drowsiness. S. Stup. = Semi-stuporose. S. Coma. = Semi-comatose. HPC = High-pitched cry. + MT = Increased muscle tone.
Column F: CSF = Cerebrospinal fluid. Prot. = Protein. (*a*) Second specimen.
Column G: (*b*) Fine crepitations at right base. (*c*) Radiological evidence of bronchopneumonia. (*d*) Clinical and radiological bronchopneumonia left lower lobe. (*e*) Crepitations at both lung bases. (*f*) Crepitations right middle lobe.
Column H: G = Good. F = Fair. P = Poor
Columns I–P: * Before treatment. † After treatment. ‡ During a relapse. †† 2-3 months later.
Column Q: S.G. = Specific gravity.
Column R: Prot. = Protein.
Column S: Gran. = Granular. Hy = Hyaline. Not exd. = Not examined. Num. = Numerous. Occ. = Occasional.

Clinical or radiological evidence of acute respiratory disease was present in 6 of the 11 patients.

The degree of dehydration on admission was assessed at from 5 to 15% of the body-weight on discharge from hospital.

On admission to hospital the serum sodium ranged from 145 to 162 mEq. per litre and the plasma-chloride from 106–148 mEq. per litre. In 4 patients the serum-potassium was less than 4·7 mEq. per litre. The carbon-dioxide combining power was markedly lowered in all cases (4·5–11·6 mEq. per litre) and the blood-urea levels were always raised (74–290 mg. per100 ml.) The serum salicylate was always less than 5 mg. per 100 ml.

Serial haematocrit estimations were carried out in 8 cases, in 4 of whom there was significant fall during rehydration, while 4 remained unchanged.

In 3 of the cases there was a history of the infant's having received fluids high in solute content during the 24 hours before admission to hospital. Case 1 had received 1,200 ml. of a skimmed-milk mixture containing protein 3·93%, and Na 20, K 50, and Cl 40 mEq. per litre. Case 3 had been given orally 900 ml. of M/6 sodium lactate (Na 167 mEq. per litre) and 1,000 ml. of a skimmed-milk formula. Case 11 had received 150 ml. of intravenous M/6 sodium lactate and been fed with 400 ml. of a half-cream milk mixture.

Only 1 of the 11 children died (case 4). The findings at the autopsy were as follows: Areas of bronchopneumonia in both lungs. Patchy mucosal congestion of the intestine. The kidneys, apart from some congestion, showed no lesions either macroscopically or microscopically. The brain and intracranial venous sinuses were macroscopically normal. Section of a costochondral junction showed evidence of rickets. Fig 1 shows the serial biochemical, haematocrit and weight changes in this case.

CASE REPORTS

Case 8

In this case we were able to observe the sequence of events which led to the development of hypertonic dehydration. The child was admitted with a 3-day history of diarrhoea and vomiting and a 2-day history of cough and rapid respiration. There was no history that excessive saline or concentrated milk-powder mixtures had been given. On examination the patient was hyperpnoeic, with normal skin turgor and a rectal temperature of 105·5°F (40·8°C). Electrolyte estimations showed a sodium level of 135 mEq. per litre with a raised chloride level of 113 mEq. per litre and a low carbon-dioxide combining power (Table II). During the first 10 hours the child refused some of the milk feeds and vomited the others. No stools were passed and only one wet

TABLE II. CASE 8. BLOOD STUDIES

	Electrolytes (a)				*Urea*	*Haemo-globin*	*Packed Cell Volume*
	Na	*K*	*HCO_3*	*Cl*	*(b)*	*(c)*	
On adm. ..	135	3·8	6·8	113	109	14·0	40%
10 hrs. ..	146	4·2	6·0	118	152	13·7	40%
2 days ..	137	3·5	18·2	102	31	10·9	31%

(*a*) mEq./litre. (*b*) mg./100ml. (*c*) g./100ml.

napkin was noted. The child then became comatose and remained hyperpnoeic, the eyes became sunken, and the rectal temperature remained high (104·5°F—40·3°C). Serum-electrolyte estimations repeated 10 hours after admission showed that the serum sodium had risen to 146 mEq. per litre and the plasma chloride to 118 mEq. per litre.

With routine treatment of the hypertonic dehydration, the serum electrolyte levels returned to normal by 48 hours after admission, coincidentally with clinical improvement.

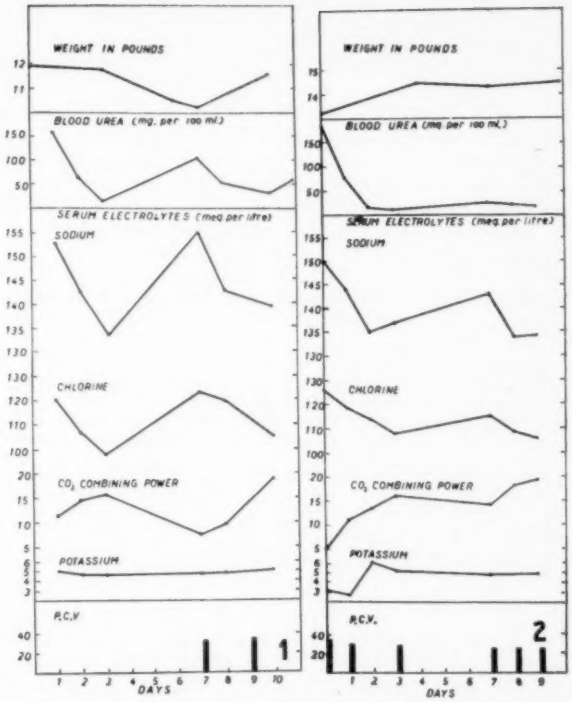

Fig. 1. Serial changes in body-weight, blood electrolytes and haematocrit in case 4 until death on the 11th day. (Note the relapse on Day 7.)

Fig. 2. Serial changes in body weight, blood electrolytes and haematocrit in case 9 throughout stay in hospital. (Note the mild relapse on the 7th day.)

Case 9

This patient was anuric for 29½ hours. When urinary output was re-established, the excretion of sodium, potassium and chloride was estimated in the urine for a period of 66 hours after commencement of treatment (Table III).

The patient, a breast-fed boy, aged 4½ months, was admitted with a history of diarrhoea for 7 days and a cough for 2 days. There was no history of ingestion of electrolyte solution. Two days before admission the infant began to breathe rapidly, and was very hot and sleepy. No urine had been passed for 24 hours.

Physical examination revealed a fairly well nourished child with marked hyperpnoea and a rectal temperature of 100°F (37·7°C). The eyes were sunken but the skin and subcutaneous tissues over the anterior abdominal wall had a doughy feel. There were crepitations audible over the right middle lobe.

Fig. 2 shows the changes in body weight, blood electrolytes and packed cell volume throughout his stay in hospital.

The patient began to pass urine 5½ hours after commencement of intravenous therapy. The first period of urine collection lasted for 42 hours. The volume passed was 750 ml. containing, per litre, sodium 48 mEq., potassium 10 mEq., and chloride 48 mEq. During the same period a total of 2,300 ml. of fluid was administered containing, in all, 59 mEq. of sodium, 34 mEq. of potassium, 68 mEq. of chloride and 25 mEq. of lactate. Urine was collected for a further 24 hours (volume 900 ml. containing, per litre, sodium 2·9 mEq., potassium 3·3 mEq., and chloride 20 m.Eq.), during which period a total of 1,900 ml. of fluid was administered containing, in all, sodium *nil*, potassium 13·5 mEq. and chloride 13·5 mEq. During the 66-hour period of rehydration the serum sodium fell from 150 to 135 m.Eq. per litre and the serum chloride from 126 to 114 mEq. per litre; while the serum potassium rose from 3·1 to 6·1 mEq. per litre.

TABLE III. CASES 9 AND 10. URINE ELECTROLYTE EXCRETIONS COMPARED WITH SERUM ELECTROLYTE CONCENTRATIONS AND FLUID INTAKE

	Serum			*Intake*				*Urine*			
	Na	*K*	*Cl*	*Vol.*	*Na*	*K*	*Cl*	*Vol.*	*Na*	*K*	*Cl*
		mEq./litre		*ml.*		*mEq.*		*ml.*		*mEq./litre*	
Case 9											
First 42 hours	144	2·7	118	2,300	59	34	68	760	48	10	48
Next 24 hours	135	6·1	114	1,900	Nil	13·5	13·5	900	2·9	3·3	20
Case 10											
First 16 hours	156	3·8	114	900	25	Nil	17	184	22	5·6	31
Next 24 hours	140	3·1	108	1,500	34	33·7	67·7	750	23	2·4	57

This patient had a mild relapse of hypertonic dehydration on the 7th hospital day caused by refusal of feeds and increased frequency of stools.

Case 10

This patient was anuric for 8 hours. Urine collected for the first 16 hours after starting treatment showed a volume (Table III) of 184 ml. containing, per litre, sodium 22 mEq., potassium 5·6 mEq. and chloride 31 mEq. The total fluid given during this period was 900 ml. containing, in all, sodium 25 mEq., lactate 8 mEq. and chloride 17 mEq. The subsequent 24-hour urine volume was 750 ml. containing, per litre, sodium 23 mEq., potassium 2·4 mEq. and chloride 57 mEq., while the amount of fluid administered during this latter period was 1,500 ml., containing, in all, sodium 34 mEq., potassium 33·7 mEq. and chloride 67·7 mEq.

DISCUSSION

The majority of previous reports have considered a serum-sodium level of 150 mEq. per litre as biochemical evidence of hypertonic dehydration. In this series, patients have been included with serum-sodium levels above 145 mEq. per litre because in several investigations at this hospital[10,11] the normal sodium values have been found to be at least 5 mEq. lower than elsewhere.

Aetiology and Pathogenesis of Hypertonic Dehydration

Balance studies[4,5] have shown fairly conclusively that water is lost in excess of sodium in hypertonic dehydration. This water deficit has been ascribed to one or more of the following factors:

1. Diarrhoeal stools of low electrolyte content.
2. Deficient water intake.
3. Increased insensible water loss, i.e. *via* lungs and skin, brought about by high fever and/or hyperpnoea[16,17] and accentuated by metabolic acidosis.
4. Ingestion of electrolyte solutions or milk feeds high in solute content.[7]

What part did the aforementioned factors play in this series of cases?

1. All of our patients suffered from diarrhoea but we were unable to determine the water and electrolyte content of the stools, and therefore could not assess the exact part played by this factor. Other authors[5,18] have found a wide variation in the electrolyte concentrations of infant diarrhoeal stools. We therefore cannot exclude the possibility of a relatively greater loss of water than of electrolyte in the stools of our infants.

2. A definite history of restricted fluid intake for some hours before admission was obtained in 4 of the cases.

3. All our patients had deep and rapid respirations (hyperpnoea) and this must have contributed materially to the water loss. In contrast, Weil and Wallace[5] noted hyperpnoea in only one of their cases with hypertonic dehydration. Because of the marked acidotic respiration with little or no signs of clinical dehydration, salicylate intoxication was considered in the differential diagnosis, but serum-salicylate levels, where estimated, were found not to be raised.

A combination of high fever and metabolic acidosis was probably responsible for the hyperpnoea in our cases. Rectal temperatures of over 102°F (39°C) were found in 7 of the 11 infants. Respiratory infection, in addition to the gastro-enteritis, may have contributed to the high temperatures and also to the hyperpnoea in 6 of the 11 cases. It is noteworthy that Finberg *et al.*[4] reported 8 cases of hypernatraemia with respiratory infection who had no diarrhoea.

Weil and Wallace[5] found a lowered blood pH in infants with hypertonic dehydration, indicating a metabolic acidosis. They also observed that plasma-chloride levels were raised relatively more than the corresponding serum-sodium levels. We have confirmed the latter observation in our patients. The hyperchloraemic acidosis results in increased rate and depth of respiration with further water loss from the lungs and establishes a vicious cycle of hyperpnoea → water loss → hyperelectrolytaemia → metabolic (hyperchloraemic) acidosis → hyperpnoea, etc.

Case 8 demonstrated well the genesis of hypertonic dehydration. The deterioration during the 10-hour period of observation could be attributed to gross hyperpnoea, high body temperature and diminished fluid intake with resultant loss of water but negligible electrolyte loss.

4. Concentrated sodium-chloride solutions or milk formulae high in solute content given during the course of diarrhoea sometimes cause hypertonic dehydration,[5,7] but this factor operated in only 3 of our cases.

The Renal Factor

The metabolic acidosis as well as the hyperelectrolytaemia are aggravated by the renal response of the infant to water deprivation. Calcagno and Rubin[19] found that in infants deprived of water there was a marked reduction in the glomerular filtration rate (GFR) and a greater tubular reabsorption of filtered sodium and chloride, with a resultant elevation of serum sodium and chloride and a low electrolyte content of the urine.[5] This contrasts with the adult undergoing water deprivation, in whom there is a normal renal plasma flow[20] and a minimal reduction of GFR.

All our patients had a markedly raised blood urea. This taken in conjunction with the frequently observed oliguria or anuria must indicate considerable reduction in the GFR.

The initial oliguria or anuria in these patients suggests that the renal lesion may be acute tubular necrosis. Against this view is the low output of electrolytes in the presence of

adequate urinary volume that was observed during the recovery phase of cases 9 and 10. There is usually a large loss of electrolytes during the diuretic phase of patients recovering from renal tubular necrosis.[21]

The Nutritional Status of our Cases

The most striking feature in this series was the good state of nutrition of the patients, 7 of them still being breast fed. Of the 11 patients, only 2 were markedly below the average weight in relation to age.[13] Furthermore, none showed any stigmata of malnutrition. Thus 81% of these children were reasonably well nourished. This contrasts with the fact that the majority (85%) of patients suffering from gastro-enteritis admitted to our wards show evidence of poor nutrition.

This factor of good nutrition in our patients assisted us in the diagnosis of hypertonic dehydration. We suspected the condition if a child with a history of diarrhoea showed marked hyperpnoea and minimal clinical signs of dehydration, and was well nourished. Nervous symptoms or signs with a high rectal temperature were the principal additional features which strengthened clinical suspicion of hypertonic dehydration.

Children suffering from malnutrition and dehydration usually have serum-sodium levels below 130 mEq. per litre, and extremely rarely is a level of 140 mEq. per litre exceeded.[22] The 11 patients reported here all had serum-sodium levels of 145 mEq. per litre or more. Thus it appears that there is some fundamental difference in the response to diarrhoea between well nourished children and malnourished infants.

Studies on the intracellular and extracellular composition of fluids in malnourished children[2,23-25] have demonstrated that even in the presence of dehydration, the water content of the cells and of the extracellular fluid is increased when compared with well nourished infants. The injection of hypertonic saline solution restores to normal the abnormal electrolyte content of the malnourished child and benefits the clinical condition. From this it follows that factors leading to hypertonicity in well nourished children will cause a return to normality in the malnourished child. This may account for our failure to find cases of hypertonic dehydration among malnourished children.

The prognosis of gastro-enteritis is better if the child is well nourished than if it is malnourished. Hypertonic dehydration, however, constitutes a serious threat even to the well noursished group as shown by comparative mortality figures of other authors.[1] In our series the mortality was low.

SUMMARY

1. The clinical and biochemical features of 11 African children with hypertonic dehydration associated with summer diarrhoea are presented. There was one death in this series and brief mention is made of the autopsy findings in this case.

2. The factors which may have contributed to the development of hypertonic dehydration in our patients are considered.

3. The pattern of electrolyte and water excretion in the urine was studied in 2 patients for periods of 40 and 66 hours after commencement of therapy.

4. This series of infants were relatively well nourished, which was in striking contrast to the majority of infants with gastro-enteritis admitted to Baragwanath Hospital, who are poorly nourished and often develop hypotonic dehydration. An explanation for the difference in response between these children and the well nourished infants, who developed hypertonic dehydration, is suggested by recent work done on the intracellular and extracellular composition of malnourished infants suffering from dehydration.

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PRAKTIESE BENADERING VAN KARSINOOM VAN DIE MAMMA*

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Karsinoom van die bors vorm omtrent een-vyfde van die maligniteite wat by vroue aangetref word. Om hierdie rede en ook omdat dit voorkom in 'n oppervlakkige orgaan wat maklik bereikbaar is, vereis dit spesiale aandag van alle praktisyns.

* Lesing gelewer by geleentheid van 'n opknappingskursus gereël deur die Nagraadse Skool-beplanningskomitee, Bloemfontein, 26 Junie 1959.

Simptome

1. Pyn kom gewoonlik nooit in vroeë gevalle voor nie.

2. Die tumor word die beste gevoel met die plat hand teen die borskaswand, veral as die M. pectoralis major aangespan word deur die pasiënt met die hand op die heup te laat druk. Hierdie tumor is gewoonlik hard, onreëlmatig, vry beweeglik, of gefikseerd.

3. Die toestand van die vel oor die tumor kan ongeskonde wees, of ingetrek by besigtiging of wanneer die vel opgelig

word. *Peau d'orange, cancer et cuirasse* en ulserasie is gewoonlik laat verskynsels.

4. Die tepel. Op die volgende tekens moet gelet word: Hoogstand, intrekking, waterige of bloederige afskeiding, en ulserasie.

5. Uitsaaiïngs vind as volg plaas:

(*a*) Direk binne in die melkbuise of buite die melkbuise.

(*b*) Deur die limfbaan na die verskillende groepe van okselkliere, na die groep van interne borskliere langs die A. mammaria interna, of oor die rectus. Verspreiding kan plaasvind na enige van hierdie groepe kliere (afgesien van waar die tumor geleë is) en selfs na die teenoorgestelde kant. Hierdie feit is van groot belang by die behandeling en prognose.

(*c*) Deur die bloedstroom. Verspreiding langs hierdie weg is gewoonlik laat, behalwe in die akute vorms, en gaan hoofsaaklik na die longe, lewer, brein en die skelet.

(*d*) Transsoelomiese uitsaaiïng is gewoonlik laat.

Differensiële Diagnose

Die volgende toestande is soms moeilik met sekerheid van karsinoom te onderskei: chroniese absesse, traumatiese vetnekrose, siste, displastiese toestande, en fibro-adenome.

Daarom is daar net een metode om 'n juiste diagnose te maak, en dit is proefeksisie. Ons oogmerk is om juis in hierdie gevalle so gou as moontlik sekerheid te kry. Ons gaan dus tot 'n biopsie en wel 'n *eksisie*-biopsie oor, d.w.s. sonder om aan die tumor self te sny, maar dit te verwyder saam met 'n rand van gesonde weefsel. Die diagnose kan dan met sekerheid mikroskopies vasgestel word.

BEHANDELING

Daar bestaan verskeie behandelingsmetodes en daar is in die laaste tyd groot vordering, veral met betrekking tot laat gevalle, gemaak.

1. *Chirurgiese verwydering:* Dit bestaan uit 'n radikale mastektomie met opruiming van die okselkliere. Sommige chirurge volstaan hiermee in die vroeë gevalle. Vanweë die feit dat in 'n sekere persentasie van gevalle, veral waar die tumor oor die mediale kwadrant voorkom, die uitsaaiïngs na die interne groep van borskliere gaan, is daar chirurge wat ook hierdie groep kliere verwyder. Dit is natuurlik 'n groot ingreep en die tyd sal leer of dit as roetine aangeneem moet word.

2. *Bestraling.* As 'n behandeling op sy eie word dit waarskynlik vandag net as 'n versagtingsmiddel in laat gevalle toegepas.

3. *Kombinasie van chirurgie en bestraling.* (*a*) Bestraling van die bors en sy dreinasie-gebied gevolg deur operasie word nie algemeen toegepas nie.

(*b*) Lokale mastektomie gevolg deur bestraling van die bors en sy dreinasie-gebied. In die hande van McWhirter gee dit net sulke goeie resultate as radikale chirurgie, maar ons moet onthou dat hy 'n baie groot ervaring het en sy tegniek perfek kon ontwikkel. In ons land bestaan nie daardie geweldige hoeveelheid materiaal nie en dit is dus moeiliker, veral in die kleinere sentra, om hierdie metode met dieselfde vertroue toe te pas.

(*c*) Radikale operasie, gevolg deur bestraling met spesiale klem op die interne borskliere en supraklaviculêre groepe van kliere, is waarskynlik onder ons omstandighede die aangewese metode van behandeling.

4. *Hormonale behandeling* het in die laaste jare sterk op die voorgrond getree. Dit het geblyk van groot nut in gevorderde gevalle te wees, d.w.s. waar uitsaaiïngs deur die liggaam reeds bestaan.

(*a*) Tot 5 jaar na die menopouse word testosteroon as metieltestosteroon na kastrasie toegedien soos volg: 10 mg. *b.d.* vir eerste week, 25 mg. *b.d.* die tweede week, en 25 mg. *t.d.s.* daarna vir ten minste 3 maande. Hierdie behandeling veroorsaak vermanlikingsimptome wat soms taamlik lastig kan wees.

(*b*) Estrogeen word vanaf 5 jaar na die menopouse as stilbestrol toegedien. Die dosis word geleidelik tot 0·6 mg. *t.d.s.* verhoog.

Wanneer die hormone sy effek begin verloor, word 'n tweesydige adrenalektomie of 'n hipofisektomie gedoen. Hierdie hormonale behandeling veroorsaak 'n groot tydelike verbetering by sommige pasiënte. Dit laat hulle 'n tydlank in betreklike gemak voortleef.

5. *Kombinasie van hormone en anti-mitotiese stowwe,* bv. mosterdgasderivate soos tiotepa, wat intramuskulêr toegedien word, skyn besliste belofte in te hou.

OPSOMMING

Die simptome, differensiële diagnose en behandeling van kanker van die bors word kortliks bespreek.

SUMMARY

The symptoms, differential diagnosis and treatment of carcinoma of the breast are discussed briefly.

FORTHCOMING INTERNATIONAL MEDICAL CONFERENCES

The Third International Congress of Physical Medicine will be held on 21 - 26 August 1960 at The Mayflower, Washington, D.C., USA. A preliminary programme may be had on request by writing to Dorothea C. Augustin, Executive Secretary, Third International Congress of Physical Medicine, 30 N. Michigan Avenue, Chicago 2, Illinois, USA.

The Seventh International Conference of the Society for Biological Rhythm will be held at Siena, Italy on 5 - 7 September 1960. The theme of the Conference is endogenous rhythms and the third day will be held in conjunction with the *International Basimetric Society*. The President of the Conference is Professor Dell Acqua of the University of Ferrara.

Applications for lectures or participation are to be sent to the Secretary of the Society, Dr. A. Sollberger, Department of Anatomy, Caroline Institute, Stockholm 60, Sweden, before 1 June 1960. In regard to lectures on basimetry, the applications should be sent to Dr. J. Wilder, 1150 Fifth Avenue, New York 28, N.Y., USA, and a copy to Dr. Sollberger. Preference will be given to lectures on endogenous biological rhythm and basimetry as well as to presentations documented by adequate experimental material and methods. Applications should be accompanied by a brief typed half-page summary. On the day of presentation a typed copy, not longer than 3 typed pages, of the actual lecture including adequate copies of figures, should be delivered to the Secretary.

Lecturing time is 45 minutes for referees and 20 minutes for lecturers. The Conference languages are English, French and German. The participation fee is 6.00 it. lire for non-members and 4.00 it. lire for members to be paid at Siena. At the Congress Bureau the Reports from the 3rd (Hamburg), 4th (Basle) and 5th (Stockholm) Conferences may be ordered. Members will receive these reports free of charge.

EDITORIAL : VAN DIE REDAKSIE

ENDOGENOUS HYPOGLYCAEMIA

Insulin has produced hypoglycaemia not only when used therapeutically in diabetics or in 'shock therapy' by the psychiatrists or in the insulin-tolerance test in the diagnosis of hypopituitarism, but also when used by the malingerer, the suicide, and the murderer. It has recently been stated[1] that tobacco-smoking is a further exogenous cause of hypoglycaemia, but one would like to see this confirmed before accepting a full causal relationship.

What may be termed 'physiological hypoglycaemia' may be found in the newborn infant. The brain at this stage can evidently withstand quite profound glucose deprivation without being in any way harmed. This applies not only to babies of diabetic mothers, although it may be that the degree and duration of the hypoglycaemia is on the whole greater in the diabetic's infant.[2]

Organic endogenous hyperinsulinism is produced by a single adenoma of pancreatic islet beta cells, by multiple adenomas, or, rarely, by a general β-cell hyperplasia. We have no knowledge of the mechanism by which the activity of the insulin-producing cells is controlled in these cases. It is probable that the secretion of the normal β cells around an adenoma is suppressed, since they show degranulation, but they recover rapidly after removal of the tumour. If this is so, then the tumour itself is presumably supplying all the insulin needed by the body. This conception could explain why a diabetic type of glucose-tolerance curve may be found in this condition. The body's anti-insulin mechanism (adrenal, etc.) would be over-stimulated by the excessive circulating insulin in the fasting state. The ingested glucose could not then evoke the normal β-cell insulinogenic response, since the tumour is non-responsive and the non-tumourous β cells are suppressed. Hyperglycaemia may then, paradoxically, appear.

Whipple[3] described the basic triad of clinical features which distinguishes this syndrome—a persistently low fasting blood sugar, cerebral disturbance in the fasting state, and relief of this disturbance by glucose. Symptoms of hypoglycaemia may, however, occur at other times, and in some cases the glucose-tolerance curve may be normal. To bring out the underlying tendency to hypoglycaemia it may be necessary to starve the patient for 24 hours and even to follow this with vigorous exertion. Any blood sugar found to be lower than 30 mg. per 100 ml. (by ordinary laboratory methods) is virtually diagnostic of pancreatic organic hyperinsulinism (except for one or two metabolic disorders in children, such as the infantile hypoglycaemosis of McQuarrie[4]).

The neurological manifestations of the severe degrees of hypoglycaemia, such as may be found in this syndrome, are very varied. Apart from the symptoms of stimulation of the sympathetic adrenalin system, which are the early features, one may see various disturbances of vision, including diplopia, squint, micropsia and macropsia; rotatory vertigo, dysphasia; hemiparesis; and gradual or sudden loss of consciousness, which may result in irreversible cerebral damage. Williams[5] claimed that motor-neurone disease with widespread muscle weakness, wasting, and fasciculation in a patient who is subject to episodes of disturbed consciousness should suggest endogenous hypoglycaemia.

The pancreatic adenomas which produce this syndrome are quite distinct from those which are associated with the tremendous hyperchlorhydria and overwhelming tendency to peptic ulceration described by Zollinger and Ellison.[6] In these cases, in which persistent watery diarrhoea and malabsorption may also occur, the adenoma arises from the α or quite possibly the γ or δ cells of the islets. However, to confuse the issue, tumours of the pituitary, active parathyroid tumours, and even pancreatic insulinomas are also sometimes found in this syndrome. Apart from the Zollinger-Ellison syndrome, multiple pancreatic insulinomas may occur in combination with pituitary tumours (usually chromophobe) and with multiple parathyroid adenomas. Plural endocrine involvement of this nature is characteristically familial.

Hypoglycaemia may occur two or more hours after meals in patients who have suffered gastrectomy, gastrojejunostomy or vagotomy. Under such conditions readily absorbable carbohydrate foodstuffs pass rapidly into the small bowel, are quickly absorbed and stimulate an excessive production of insulin, leading to a 'reactive' hypoglycaemia. There is typically a latent period of many months after operation before the symptoms appear.

In the so-called 'functional' hypoglycaemia the mechanism is believed to be similar—that is to say, in certain people, who are frequently neurotic, gastro-intestinal hurry and over-rapid glucose absorption develop, leading to hypoglycaemic symptoms several hours after a meal. Because of this, symptoms are not found in the early morning fasting state—an important differential point. The appreciation of the likelihood of hypoglycaemia as the cause of symptoms in these people is rendered difficult because a low blood sugar may itself produce both emotional and somatic features resembling those of neurosis.

The treatment of the patient with reactive or functional hypoglycaemia is to let him have frequent small meals, high in protein content. Protein is more slowly absorbed and produces a more gradual and sustained rise in blood sugar with a lower peak level, and so prevents rather than provokes a delayed hypoglycaemic response. Fat is also allowed in the diet dependent upon the total caloric requirement, but carbohydrates are severely restricted. Incidentally this same principle is used in the dieting of obese patients—a high protein breakfast will prevent the hypoglycaemic hunger of the mid-morning and so help to obviate between-meal nibbling.

1. Berry, M. G. (1959): Ann. Intern. Med., **50**, 1149.
2. Farquhar, J. W. (1956): Arch. Dis. Childh., **31**, 203.
3. Whipple, A. O. (1938): J. int. Chir., **3**, 237.
4. McQuarrie, I. (1954): Amer. J. Dis. Child., **87**, 399.
5. Williams, C. J. (1955): Brit. Med. J., **1**, 707.
6. Zollinger, R. M. and Ellison, E. H. (1955): Ann. Surg., **142**, 709.

DIE PROBLEEM VAN OORBEVOLKING

'n Interessante en ironiese lig word gewerp op aspekte van die vraagstukke van voorbehoedende medisyne en gemeenskapsbeplanning deur die feit dat kunsmatige bevrugting *en* die gebruik van voorbehoedmiddels op groot skaal deesdae albei as dringende probleme beskou word. Dat dit so is kan 'n mens verstaan, en daar hoef nie noodwendig 'n teenstelling te wees tussen hierdie twee probleemgesteldhede nie.

Aan die een kant is dit 'n feit dat daar baie kinderlose egpare is wat hulle na die mediese wetenskap wend in die verwagting dat iets moontlik gedoen sal kan word om hulle uit hulle toestand van ontbering, eensaamheid, gemis, en 'n gevoel van andersheid te verlos. In terme van menslike welstand en geluk is dit 'n probleem wat sonder twyfel deur die geneesheer aangepak moet word.

Aan die anderkant is dit egter ook waar dat geen intelligente en denkende mens sy oë kan sluit vir die dreigende gevare van onbeheerde en onbeteuelde bevolkingsaanwas nie. Dat ons hier voor 'n moeilike en ingewikkelde probleem staan, ly geen twyfel nie. En, ons is wel bewus van die bestaan van dieperliggende morele en godsdienstige implikasies van 'n doelbewuste beleid van gesins- en gemeenskapsbeplanning op hierdie gebied. Tog kan ons ons nie losmaak nie van die spesifiek-professionele en algemeen-menslike verpligting wat op ons rus om aan die wêreld te dink, nie net soos hy vandag is nie, maar ook soos hy na verwagting in die toekoms daar sal uitsien. En, omdat die vraagstuk van oorbevolking van die wêreld so nou saamhang met die vraagstukke van voedselvoorsiening en van menslike aanpassing in die algemeen, word die probleem van bevolkingsaanwas by uitstek 'n uitdaging vir die geneesheer.

Dit is nie moontlik om in die kort bestek van hierdie artikel reg te laat geskied aan al die fasette van hierdie ingewikkelde probleem nie. Nogtans sal dit goed wees om na die belangrikste aspekte van die probleem te verwys in die lig van historiese en praktiese verwysings.

Sedert die dae van Malthus is die vraagstuk van onproporsionele bevolkingsgroei (met betrekking tot voedselvoorsiening en verwante behoeftes) al druk bespreek. Die essensie van Malthus se teorie sou kon opgesom word deur te sê dat bevolkingsgroei nie in dieselfde verhouding as dié van die aarde se vermoë om te voorsien in onderhoudsbehoeftes, toeneem nie.

Na die eerste formulering van Malthus se teorie in 1798, is hierdie probleem bespreek in ekonomiese, politieke, literêre, filosofiese, godsdienstige en geneeskundige kringe, en heftige stryde het om die vraagstuk ontwikkel. Alhoewel almal nie oor die saak saamstem nie, word dit vandag in elk geval aanvaar dat die kwessie van moontlike oorbevolking wel 'n dringende probleemgesteldheid is. Breedvoerige planne vir die toepassing van geboortebeperking is byvoorbeeld al as praktiese beleid in verskillende lande toegepas.

So 'n omvangryke program vir geboortebeperking is onder andere in Indië toegepas. Weliswaar het Indië nog nie juis daarin geslaag om sy bevolkingsgroei te verminder nie. Maar, Indië het nou begin om vrywillige sterilisasie van vroue met meer as drie of vier kinders te oorweeg. In Japan het die geboortesyfer gedurende die afgelope tien jaar met nagenoeg die helfte gedaal—maar in Japan is die metode van wettige vrugafdrywing gebruik wat in meeste Westerse lande nie aanvaar sal word nie. In sommige van die Westerse lande, soos in Engeland, byvoorbeeld, is daar 'n stadige toename in die bevolking.

Die feite bly egter nog staan dat die V.V.O. sy skatting van die bevolking van die wêreld moes hersien nadat dit bekend geword het dat Sjina een honderd miljoen meer mense het as wat verwag is. Ook word dit op grond van die skatting van deskundiges bereken dat die bevolking van die wêreld binne die volgende veertig jaar sal verdubbel as die teenwoordige aanwas voortduur.

Dit is nie vir ons moontlik om hier 'n oplossing vir hierdie moeilike en belangrike vraagstuk te gee nie. Wat egter wel van belang is, is dat dit al meer besef word dat ons hier met 'n vraagstuk te doen het wat sowel spesifiek-mediese as algemeen-menslike implikasies het, en dat dit 'n vraagstuk is waaraan alle verantwoordelike persone aandag behoort te skenk. Vir die voorbehoedende medisyne wag hier 'n groot uitdaging.

UNUSUAL ACCIDENTS AND INCIDENTS OF ANAESTHESIA*

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Any anaesthetist of long experience will bear testimony to the fact that when accidents or incidents occur during or after the administration of an anaesthetic, they invariably occur with dramatic suddenness. However careful and assiduous the anaesthetist might be, the unexpected or seemingly inexplicable accident or incident will crop up from time to time. Only recently, in an interesting publication,[1] Keating has comprehensively reviewed the causes and has endeavoured to indicate the possible prevention and treatment of these distressing accidents of anaesthesia.

Covering a period of 25 years, some of the episodes recorded here might well be regarded as being of academic interest only, because the probability of a similar occurrence in one's own practice may be remote or virtually non-existent; but the mere fact that they have occurred is nevertheless of some interest and may be of some importance.

1. *Ether Explosion Caused by a Short-circuit*

An ether explosion at the New Somerset Hospital in 1937[2] brought home very vividly to me what a hazardous occupation anaesthesia as a speciality might be! This episode took place whilst we were engaged on the sixth tonsillectomy on a very hot and oppressive summer's day. The temperature recorded at the Royal Observatory was 80°F and the relative humidity 70°. The theatre was poorly ventilated, the temperature inside being well above 80°F. Anaesthesia was induced with ethyl chloride followed by ether on the open mask, and the anaesthetic was maintained with the Shipway apparatus.

The explosion occurred dramatically just as the surgeon was about to commence the operation. The flame appeared

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to have travelled along the tube leading to the Shipway apparatus, which caught fire and blew up with a roar that shook the old-fashioned turreted New Somerset Hospital (built in 1856) to its very foundations. Miraculously the patient escaped injury. I was not quite so fortunate, for I was concussed by the blast, and received a 2-inch gash and glass embedded in my scalp.

On subsequent examination of the electrical equipment it was found that the lamp which reflected light on to the surgeon's head-mirror had developed a 'short' and, having been switched on for 2½ hours, had overheated. Incidentally, the atmosphere in the theatre was heavily laden with ether.

In view of the fact that the flame appeared to have started not in the patient's mouth, but at some point along the course of the tube leading from the Boyle-Davis gag to the Shipway apparatus, it must be assumed that a leak in the tube had produced a sufficient concentration of ether near the hot lamp for ignition to take place, followed by a chain reaction, the explosion proceeding to the hole in the tube and thence to the whole apparatus. The 'short' in the lamp may conceivably have caused an arc and produced a higher temperature, which would make this particular mechanism more probable.[3] In view of the high humidity, static electricity has been ruled out as a causal factor.

2. *Decomposition of Ether*

Opinions tend to differ on whether decomposition of ether is of clinical significance, but it now appears to be an accepted fact that unpleasant after-effects can be and are produced by the use of deteriorated ether.[4] Coste[5] has come to the conclusion that peroxides themselves are not the cause of these after-effects and that some undertermined substance, of which the presence of peroxides might be an index, may well be the cause. Wesley Bourne[6] comes to the following conclusions, viz. that up to 0·5% of acetaldehyde does not produce any significant changes, that ether peroxide can cause a lowering of blood pressure and pronounced respiratory disturbances, and that ethyl mercaptan, although a foul substance, does not have much influence up to 1%.

Symptoms simulating shock. In March 1946 the nursing staff at the Wynberg Military Hospital reported to me that post-operatively many of our patients were behaving 'peculiarly'. We had 3 anaesthetists on our staff at that time and the following complications were noticed by us all, viz. (1) a general tendency to profuse sweating on return to the wards and for several hours afterwards; (2) respiratory depression; (3) cyanosis lasting up to an hour or longer; and (4) prolongation of recovery time. Two patients had developed severe hypotension during the course of a tonsillectomy and appendicectomy respectively. The anaesthetic commonly used at that time was a sequence of pentothal, nitrous oxide, oxygen, and ether. There was no obvious causal factor, apart from the ether, since patients to whom pentothal, nitrous oxide and oxygen had been administered showed no untoward symptoms. Various samples were therefore sent to the Senior Government Analyst, who reported as follows: 'The samples do not comply with the standards for Aether Anaestheticus as laid down in the B.P.C. of 1932. The distilling ranges are all too high and all samples contain more or less excessive quantities of peroxides, aldehydes and acetone'. In view of this adverse report, the entire batch of ether was condemned by the D.G.M.S. and a fresh brand of ether substituted. This was followed by an immediate and total disappearance of all post-operative sequelae.

A cockroach in the ether. In August 1957, during the administration of two consecutive anaesthetics at a private nursing home, a peculiar unpleasant odour in the theatre was the subject of much comment and perplexity. It was similar to the smell of putrefying red-bait, well known to fishermen at the Cape. Both patients exhibited identical post-operative symptoms, viz. (1) profuse sweating, (2) excessive vomiting, and (3) marked pallor. Next day, at the same nursing home, I opened a sealed container of ether and, on pouring the contents into the ether bottle on the Boyle's machine, was amazed to see a cockroach suddenly appear, surrounded by an oily exudate the size of a two-shilling piece. In view of these two strange episodes, it was decided to send several bottles of ether for analysis. The Government analyst reported that (*a*) acetone and aldehydes were present, (*b*) tests for peroxides were negative, and (*c*) the residue on evaporation was *excessively high*, viz. 9, 19 and 58 mg., indicating the presence of an impurity (B.P.C. specification—not more than 1 mg.). The foul putrefying odour was identified as being probably due to ethyl mercaptan.[7] None of the samples submitted complied with the 1953 B.P.C. specification.

3. *Methylated Spirit mixed inadvertently with Ether*

During the administration of a sequence of pentothal nitrous oxide, oxygen, and ether, at a well-known teaching hospital, I was struck by the fact that the induction was uneven and unduly protracted; some second instinct made me take a sniff at the ether bottle on the Boyle's machine and to my surprise, mixed with the smell of ether, was a strong smell of methylated spirit.

Close investigation and inquiry elicited the information that white methylated spirit for cleansing purposes had been poured into an empty ether bottle, but had not been labelled as such. The 'anaesthetic nurse' had used this bottle in error to 'top-up' the ether pot in the Boyle's machine

Post-operatively the patient showed amusing signs of inebriation. He was kept under careful observation for some days, since it is known that methyl alcohol sometimes impairs vision by the development of an optic neuritis. Fortunately no complications ensued.

4. *Post-operative Cerebral Oedema*

This patient, aged 38, who was hypertensive (blood pressure 240/130 mm. Hg), had left the theatre at the termination of a hysterectomy in seemingly good condition—reflexes very active, almost, but not quite, awake. There had been a steady drop in blood pressure during the operation from 180 to 140 mm. Hg. Seen in the ward about ½ hour later, the patient was restless, confused and agitated, and presented the clinical symptoms of cerebral irritability. A diagnosis of cerebral oedema was considered, due possibly to one or other of the following two causes:

1. *Mechanical*. A steep Trendelenberg position[11] in a hypertensive patient.

2. *Anoxia*. The anaesthetic used had been pentothal, nitrous oxide, and oxygen and intermittent dilute scoline, with controlled and occasionally assisted respiration. A slow steady drop in pressure, plus the very steep Trendelenberg position and possibly inadequate assisted respiration, may well have produced a slight degree of undetected anoxia, with the resultant development of cerebral oedema.

An intravenous injection of 50 c.c. of 50% sucrose[12] brought about an immediate dramatic cessation of all symptoms and the patient became cooperative and comprehending. Subsequent recovery was uneventful.

Other Incidents

5. *Broken Intravenous Needles.* Two cases are recorded of an intravenous needle being snapped during the administration of pentothal by a sudden movement of the patient's arm. In both the nurse had been specifically instructed to hold the arm in case of any movement, and in both the nurse's attention had been momentarily diverted. In the first case the needle had to be removed subsequently under the screen, which proved to be a very difficult and protracted operation. In the other case the needle was located and removed within a few minutes of the mishap. The importance of tying down the arm during the administration of pentothal should be strongly emphasized.

6. *Granuloma of the Vocal Cord following Intubation.* After a tonsillectomy, carried out under endotracheal anaesthesia, the patient became hoarse and this hoarseness persisted. Subsequent laryngeal examination by the surgeon concerned revealed a granuloma of the cord.

7. *Obstruction due to Impaction of a small Dental Plate.* A small dental plate containing two teeth, which had not been removed pre-operatively, became dislodged during the recovery period, impacted over the cords, and very nearly caused a fatality.

8. *Persisting Tracheal Irritation caused by a Gauze Thread from an Oral Pack.* After an operation performed under endotracheal anaesthesia, the patient persisted in complaining intermittently of 'something irritating her throat'; this was accompanied by an irritant cough. On the 18th day she suddenly coughed up a thread of gauze about $1\frac{1}{4}$ inches long, which had obviously become loosened from the oral pack and had found its way down the trachea. It had become semi-rigid, acting as a chronic irritant.

9. *Nasal Endotracheal Tube slipping down into the Post-nasal Space.* About 10 years ago, at the completion of a thoracoplasty in which the patient had been intubated nasally because oral intubation had been technically difficult, I disconnected the Rowbotham connection in order to apply suction to the tube before removal. The patient's reflexes were much brisker than I had bargained for and, in the brief moment which elapsed whilst turning round to get the suction, the tube disappeared from view into the post-nasal space, impinging on the carina and initiating a very severe laryngeal and bronchial spasm; the patient's teeth were tightly clenched and oxygen under pressure failed to overcome the anoxia. 5 c.c. of pentothal was injected rapidly and the jaw relaxed sufficiently to enable me to extract the tube *via* the mouth and inflate the lungs with oxygen. Incidentally, scoline was not available at that time.

10. *Persistent Extubation Spasm after use of Scoline.* Pentothal-scoline sequence was used for a bronchoscopy, and after removal of the bronchoscope, as soon as the effects of the scoline started to wear off, a dangerously severe laryngospasm supervened. More scoline was given to overcome the spasm, but each time the effects of the scoline wore off a vicious circle of acute laryngospasm and anoxia supervened. Eventually nitrous oxide and deep ether anaesthesia successfully terminated the spasm.

11. *Cardiac Irregularity caused by an inflated Endotracheal Tube in a Patient under Light Anaesthesia.*[8,9] At the termination of an operation for excision of a pleurocutaneous fistula and decortication, the pulse suddenly slowed from 64 to 38 per minute and became irregular. At this stage the level of anaesthesia was very light and the reflexes were very active. *Prostigmin had not been used.* Deflation of the cuff caused a dramatic change—the pulse speeded up to 72 and the rhythm became perfectly regular.

12. *Fatal Air Embolism during Utero-tubal Insufflation.* A sad tragedy occurred suddenly during the course of a utero-tubal insufflation. The patient, who had been anaesthetized with pentothal, nitrous oxide, and oxygen, suddenly gave a few rapid gasps and cardiac failure supervened immediately afterwards. The surgeon had insufflated air instead of carbon dioxide and this was undoubtedly the cause of the fatality.[10] The post-mortem findings were as follows: (1) Frothy blood in the chambers of the right side of the heart and in the great veins and their tributaries; (2) air in the right parametrial tissues and neighbouring retroperitoneal haemorrhage; (3) death due to acute circulatory failure from air embolism.

13. *Fatal Spontaneous Perforation of an Atheromatous Ulcer in the Right External Iliac Artery.* This extraordinary episode occurred at University College Hospital, London, in 1939. A patient aged 59, to whom I was administering an anaesthetic for a cystoscopy, suddenly showed signs of impending peripheral failure. The patient was removed to the ward where, in spite of resuscitative measures, death occurred some 3 hours later. The post-mortem, performed by Prof. G. Cameron, revealed that death was due to a spontaneous perforation of an ulcer in the right external iliac artery. The full report reads as follows: 'The right external iliac artery showed a long deep wrinkle on its outer margin with a small perforation about 3 mm. in length amongst some atheromatous ulceration. This communicated with a huge retroperitoneal and pelvic haematoma, which had tracked along the right ureter to the pelvis of the right kidney and to the anterior abdominal wall, and also along the route of the mesentery, through which it had burst, so that the abdominal cavity contained about 40 ounces of free blood.'

14. *Self-asphyxia in an Obese Patient.* A huge, obese, bull-necked individual underwent a haemorrhoidectomy at 4 p.m. The anaesthetic was uneventful and had been combined with a local infiltration. The patient left the theatre a 5 p.m. and within a few minutes was awake and talking. At 7 p.m. $\frac{1}{4}$ gr. of morphine was administered and at 11 p.m. the night sister reported that he was fast asleep and snoring heavily. When revisited at 2.30 a.m. he was dead. At the special request of the relatives no post-mortem was held, but the opinion expressed by the Government pathologist was that the appearance of the body was consistent with that of asphyxia. Naturally the question of a coronary thrombosis or a cerebral vascular accident was at first uppermost in one's mind as the cause of death. The patient's wife then volunteered the information that even during normal sleep her husband would frequently 'frighten the life out of her' by going very blue, struggling for breath whilst breathing very stertorously. It is quite possible that the patient's absorption of morphia occurred very slowly and as he was bull-necked and edentulous a slow chronic anoxia supervened during the night, caused by obstruction of the jaw, finally terminating in cardiac failure.

15. *Radial Nerve Palsy caused by Pressure of the Operating-Table Screen.* A very obese middle-aged patient, after an emergency appendicectomy, developed a left wrist drop and months of physiotherapy were required before movement and sensation returned. The cause of this unfortunate accident was pressure over the left musculo-spiral nerve, the left arm having been compressed between the screen of the operating table and the patient's body.

16. *Post-anaesthetic Aphasia.* A male patient aged 51, normotensive, underwent lower lobectomy in 1948 for a carcinoma of the left lung. The anaesthetic then used was pentothal-cyclopropane. On waking up the patient was found to be aphasic. The neurologist reported as follows: 'This is almost certainly a vascular lesion of either embolic or thrombotic type, which must have occurred at the time of the operation or immediately thereafter.' The only unusual episode during the operation was an unexplained rise in blood pressure.

17. *An Overdosage with Cyclopropane* was caused by a nurse accidentally brushing against the cyclopropane cylinder key. The Boyle's machine used at that time had no fine adjustment valve.

18. *Patient falling off the Operating Table.* At the completion of a tonsillectomy an adult patient was turned on his side after having been entrusted to the staff nurse for transference to the trolley. The nurse unfortunately had her attention distracted for a few seconds, during which time the patient suddenly vomited, heaved, and overbalanced, crashing to the theatre floor with a sickening thud. Very fortunately, apart from extensive bruising, no serious injury was sustained.

19. *Postural Cerebral Anaemia.* After pneumonectomy the patient left the theatre at 12.35 p.m., reflexes very brisk. On being returned to the ward she was placed bolt upright and her level of unconsciousness, according to the sister, appeared to deepen. When seen at 4.45 p.m. she had apparently not yet regained consciousness. I ordered her to be placed flat in the bed and gave her a few whiffs of carbon dioxide, and within 2 minutes she was wide awake and able to converse perfectly normally with her husband, who had been waiting anxiously at the door of the ward for some hours.

20. *A Fracture-dislocation of the Metacarpo-phalangeal Joint of the Right Thumb* was sustained by the writer whilst using his hand to open the key of an oxygen cylinder.

21. *Inadvertent Injection of Sterile Water instead of Pentothal*; and the patient wondered why she had not fallen asleep after counting ten!

22. *Dislocation of Jaw.* After having received 4 c.c. of pentothal during induction, the patient, a very husky male, gave a tremendous yawn and dislocated his jaw! More pentothal was given and the dislocation reduced.

23. *Loosened Suction Tip.* A missing suction tip was found in the patient's mouth after a submucous resection of the nose; it had inadvertently slipped off during post-operative toilet of the post-nasal space.

24. *Trauma to the Right Pillar of the Fauces necessitating Suturing.* This episode occurred during a technically difficult oral intubation, in the days long before scoline and the Macintosh laryngoscope were in general use. The right pillar was lacerated and several stitches were inserted to control the persistent ooze.

CONCLUSION

In reviewing this series of mishaps in retrospect, it is evident that whilst certain accidents of anaesthesia occur spontaneously and are unavoidable, some are preventable and in the final instance the anaesthetist should trust nobody but himself. Our watchwords should be 'Constant, diligent vigilance.'

SUMMARY

When accidents or incidents occur during or after the administration of an anaesthetic, they invariably occur with dramatic suddenness. However careful or assiduous an anaesthetist might be, the seemingly unexpected or inexplicable accident or incident will crop up from time to time.

The episodes described in this paper cover a period of 25 years.

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SECRETIONAL ANOXIA AND TRACHEOTOMY*

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The operation of tracheotomy was first mentioned by Galen and Areteus in the 2nd and 3rd centuries A.D. but they did not use the term tracheotomy.[1] Galen used a word meaning to cut the larynx or artery—he used artery, arteria aspera, and bronchus, all to mean the windpipe. Subsequently the terms laryngotomy and bronchotomy were used more or less indiscriminately by various writers, and it was Fabricius in the 16th century who was first to use the term tracheotomy. However, the terms laryngotomy and bronchotomy continued to be used by many until the 19th century, when Bretonneau and Trousseau firmly established the operation as a life-saving procedure in diphtheria and also firmly established the present name. The first tracheotomies and, for that matter, tracheotomies up until recent times were performed almost exclusively for obstructive laryngeal lesions of various aetiology.

With the development of the concept of secretional anoxia the role of the operation has been greatly extended. Echols *et al.*[2] report that, out of a total of 108 tracheotomies performed in a general hospital between January 1947 and July 1950, only 39 were for obstructive lesions, while 69 were for the prevention or treatment of respiratory complications. Between January and June of this year 45 tracheotomies were done by the Ear Nose and Throat Department of Groote

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Schuur Hospital, Cape Town; of these only 14 were for obstructive lesions, while 31 were for the management of secretional obstruction and allied problems.

The ear nose and throat surgeon has thus become an integral part of the team in the management of this type of respiratory insufficiency.

The present-day role of tracheotomy owes much to the work of those concerned in the management of respiratory problems in poliomyelitis. With the recognition of the place of the operation in poliomyelitis it was logically inevitable that the indications would be widened to include a variety of disease states with similar respiratory problems. Wilson,[3] of Harvard, in 1931 recognized the relief afforded by direct removal of secretions from the respiratory tract. He considered tracheotomy a help in selected cases although a radical procedure. More experience convinced him, according to reports in 1941 and 1943, that sometimes tracheotomy was life-saving. More thought and study were given to the problem after Galloway[4] published an article in 1943 in whch he observed that tracheotomy had saved 2 poliomyelitis patients who otherwise would have succumbed to the disease. Epidemics in succeeding years gave American physicians more opportunity to recognize the respiratory obstruction early, when formerly it was frequently overlooked. Priest *et al.*[5] reported thus on the 1946 Minneapolis epidemic: 'Of 75 patients on whom tracheotomy was performed 29 survived and 19 of these left little doubt that tracheotomy directly saved their lives.' Reports such as these and subsequently others, notably those by Professor Lassen,[6] of Copenhagen, firmly established the place of tracheotomy in poliomyelitis and laid the foundations for further development.

Pathology of Secretional Anoxia

Normally between 1200 and 1500 c.c. of saliva is secreted daily by the average adult, and about 900 c.c. of fluid is produced in the respiratory tree. Over and above this it has been suggested that in conditions such as poliomyelitis actual hypersecretion of saliva takes place owing to autonomic dysfunction possibly caused by central stimulation of the salivary nuclei in the medulla due to anoxia or inflammation. In addition oedema of the lung and tracheobronchial inflammation produce increased fluid. Secretional obstruction is likely if (*a*) secretions, drink, food, vomit or blood spill over into the trachea because of disturbance of the swallowing mechanism, or (*b*) the patient is unable to expel secretions or inhaled material from the lungs because of the inability to cough. Either or both of these two grave disabilities may arise in a number of pathological conditions, viz., (i) acute poliomyelitis, (ii) polyneuritis of the Guillain-Barré and Landry type, (iii) coma of any aetiology, (iv) head injuries, (v) tetanus, and (vi) severe injury to the chest, neck, mouth, pharynx and larynx. Clinical observation warrants the conclusion that the immediate cause of death in many of these patients is progressive anoxia and accumulation of carbon dioxide from obstruction of the airway by mucus and other secretions and the resulting pulmonary complications—atelectasis and pneumonia. The clinical signs and symptoms of obstructed or inadequate respiration may be summarized as follows:

(*a*) A sustained diastolic and systolic hypertension due to the raised blood carbon dioxide.

(*b*) Apprehension and anxiety.

(*c*) Mental aberration.

(*d*) Signs of retained secretions in the tracheobronchial tree.

(*e*) Fever and tachycardia.

(*f*) Cyanosis, which, it should be observed, is a manifestation that should not be allowed to develop. Regardless of the disease entity, tracheotomy may be said to be indicated whenever this procedure alone will establish and maintain an adequate airway fror proper gaseous respiratory exchange.

Advantages and Disadvantages of Tracheotomy

The advantages are as follows:

1. It provides a free airway and at the same time, if necessary, the use of a cuffed tube prevents the entry of fluids and solids from the pharynx into the trachea.
2. Tracheobronchial toilet is greatly facilitated.
3. It makes it possible to use intratracheal positive-pressure respiration when indicated.

The disadvantages are the following:

1. Highly trained and experienced personnel are essential, particularly in cases requiring assisted respiration.
2. The weaning period may be difficult in patients making a poor respiratory recovery in poliomyelitis.
3. The tracheotomy tube in itself increases secretion and, if the inhaled air is not adequately humidified, viscous secretions and crust formation are likely.
4. Infection may be introduced by the repeated suction if the suction technique is not kept as aseptic as possible.
5. Lassen reported 3 cases of tracheal stenosis in 250 cases of bulbospinal poliomyelitis treated by tracheotomy and intermittent-positive-pressure respiration.

Tracheotomy Technique

The majority of tracheotomies of this type can be performed under local anaesthesia and, of course, in unconscious patients without any anesthetic at all. General anaesthesia is preferred in the following 2 groups:

(*a*) In bulbospinal poliomyelitis cases, where the operation is being done to institute intermittent-positive-pressure respiration. These patients require assisted respiration, and many of them are mentally alert, distressed and anxious; tracheotomy is then less harrowing under general anaesthesia. For spinal cases in a respirator who require tracheotomy because of supervening bulbar paralysis, the operation is much more easily done with endotracheal anaesthesia and assisted respiration outside the respirator.

(*b*) Neonatal tetanus tracheotomies are also best done under general anaesthetic with endotracheal intubation.

The tracheotomy incision will depend on the personal preference of the operator. Those who favour the transverse incision claim better cosmetic results. In fact, the cosmetic deformity of any tracheotomy is the central puckered scar, and this may follow either the vertical or the transverse incision; its size and unsightliness depend on the duration of the tracheotomy and the degree of wound infection. The vertical incision is quicker and easier as regards haemostasis, and is, for the occasional operator at least, the one of choice.

Lassen and his associates recommend a high tracheotomy in cases for intermittent-positive-pressure respiration. This is to obviate the danger of the cuffed tube slipping down one or other main bronchus. In his 1952 series of 250 tracheotomies only 3% of the patients developed tracheal stenosis and, considering that many of these patients had a cuffed tube *in situ* for anything from 3 to 6 weeks, it is difficult to know

whether a low tracheotomy would have produced better figures. I performed a low tracheotomy on the first bulbospinal case to be treated by Lassen's method in the 1956 Cape Town polio epidemic and I must confess the tendency of the tube to slip down the right bronchus required constant watching. The right-angled tube designed by Crampton Smith eliminates this hazard, however, and with it I think this objection to a below-isthmus tracheotomy falls away.

If tracheotomy is being performed with a view to intermittent-positive-pressure respiration it is necessary to excise a window from the anterior tracheal wall. This should be oval in shape with its greatest diameter in the axis of the trachea. If the tracheotomy is for an obstructive lesion or for tracheobronchial toilet only, the excision of such a window would appear to be optional, and Mosher's cruciate incision or a simple vertical division of 2 or 3 tracheal rings is used.

Management of Patient

The following points are stressed:

(*a*) Secretions should be kept as fluid as possible by adequate humidification, alevaire therapy, or full hydration. When secretions are viscid and difficult to clear, preparations such as alevaire, tryptar and pancreatic desoxyribonuclease may be of help. Galloway[7] recommends irrigation with 1-5 ml. of normal saline and 3% sodium bicarbonate solution occasionally in preference to the above agents. If treatment is adequate bronchoscopy should rarely be necessary.

(*b*) If a cuffed tube is in use this should be deflated for a minute or two every 4 hours. Before the cuff is deflated the bed should be tilted and the stomach and pharynx aspirated.

(*c*) Suction technique should be as aseptic as possible to minimize infection.

(*d*) Regular turning, percussion and chest squeezing combined with gravity and suction are important in the prevention and management of pulmonary complications.

Detubation

The following criteria are given for closure of the tracheotomy:

1. There must be complete return of swallowing.
2. The laryngeal and chest muscles must permit an adequately forceful cough.
3. The laryngeal muscles must permit maintenance of an adequate airway.
4. The patient must be able to oxygenate adequately without assisted respiration.
5. There must be no evidence of any pulmonary complication indicating bronchial obstruction and atelectasis.

The difficulty in detubation which sometimes arises in infants who have had a tracheotomy for any length of time is a familiar problem. In the first instance a laryngoscopy and bronchoscopy should be carried out to ascertain whether there is any stenosis or whether granulations are present in the trachea. When such granulations have been encountered they have, in my experience, invariably been found to be prolapsing into the trachea from the margins of the stoma and not arising within the trachea itself. I have found that the most effective way of dealing with these is to remove them under endotracheal anaesthesia with a Hartman's granulation-tissue forceps through a Welsh Alleyn operating auriscope inserted into the tracheostome. In some cases neither stenosis nor granulations are found to account for the difficulty in detubation, and I should like to submit the following explanation for this group. The infant trachea can tolerate very little reduction of its lumen without seriously impairing its airway. With the removal of a tracheotomy tube which has been *in situ* for any length of time there is an established gap in the anterior tracheal wall and therefore some degree of prolapse of soft tissue into the trachea, which is further aggravated by inspiration through the tracheostome. The infant becomes distressed and the tube has to be replaced. The only treatment for these cases is to reduce the size of the tracheotomy tube to the smallest possible, and then wait for the trachea to acquire the necessary extra capacity required to compensate for the prolapse described above.

Conclusion

In view of the team-work and trained personnel required to obtain the best results in the management of these various types of respiratory insufficiency I think it is essential to provide specific centralized units in all our major hospitals.

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A RARE COMBINATION OF TWO UNCOMMON FRACTURES OF THE CARPAL BONES

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By direct violence any one or more of the carpal bones may be fractured. Certain of them, such as the carpal navicular and lunate, are susceptible to fracture by indirect violence, and others are uncommonly or rarely damaged in this way. Among the rare carpal fractures that have been described are those of the os multangulum minus,[1,2] the os multangulum majus,[3-10] the os capitatum[4] and the os hamatum.[11-13] This paper presents the rare occurrence of a fracture of the os triquetrum and a fracture of the os multangulum majus in the same wrist at the same time.

In 1911 Deneke[14] published 3 cases of fracture of the os triquetrum and all were associated with a fracture of the styloid process of the ulnar bone. Subsequently a number of other writers described fractures of the os triquetrum.[15-23] Simon,[16] in 1928, emphasized a relationship between fractures of the triquetral bone and injuries of the navicular and lunate bones.

In 1942 Greening[24] was able to collect 20 fractures of the triquetral from 580 cases of injury to the wrist bones seen over a 2-year period in his hospital. Of his 20 cases 3 had

associated fractures of the lower end of the radius. He remarked that the observation of previous authors upon the rarity of the isolated fracture of the triquetral bone was not accurate. Fairbank[25] had 8 cases in his experience.

In 1945 Kellam and McGoey[26] reported on 192 cases of fractures and dislocations of the carpal bones. They list 175 fractures, the frequency being as follows: Os naviculare (145 cases), os triquetrum (11), os multangulum majus (4), os capitatum (3), os lunatum (2), os pisiforme (1), os multangulum minus (1), multiple (8 cases). Their list also shows that 10% of cases referred for injury to the distal third of the radius had also suffered injury to a carpal bone; and that of 331 successive cases of fracture of the radius at all levels 7% had also a carpal injury.

In 4 years in a busy out-patient department Bartone and Grieco[27] (1956) found 46 cases of fracture of the triquetral bone. Though they came to regard it as a rather infrequent carpal fracture, yet in their experience it was the second commonest fracture of the carpal bones. In 14 of their 46 cases triquetral fracture was combined with a fracture of the radius (9), the ulna (3), the navicular (2) and the hamate (1). There was no triquetral fracture associated with a fracture of the os multangulum majus. No explanation was offered for the manner of production of the combined fractures.

Classification

Bartone and Grieco[27] divide the kinds of fracture of the triquetral bone into 'dorsal chip', 'body', and 'body and dorsal chip'. There was no separation of the fragments in those fractures involving the body alone (1 case) or body and dorsal chip (8 cases). The commonest type was a fracture producing a dorsal chip. Of these there were 23 in their series of 46 cases. Bonnin and Greening[28] divide fractures of the os triquetrum into those associated with the posterior tubercle of the triquetral and involving marginal, styloid and other fractures of the distal end of the radius, those with a fracture of the posterior pole of the lunate, those with a fracture of the tip of the ulnar styloid process, and perilunate dislocation of the wrist with a fracture of the proximal pole of the navicular. They make a second group in which fractures in related bones are associated with a compression fracture of the triquetral bone. Greening[24] clearly defined 3 types of fracture of the os triquetrum as demonstrated by radiographic examination, which showed up (1) fractures extending through two-thirds of the bone at its dorsal aspect, with radiating cracks or fissures though the bone; (2) a type in which a small flake of bone is avulsed from the dorsum with considerable separation; and (3) a type in which there is a small flake with little or no separation.

Radiography

Greening[24] and Fairbank[25] state definitely that an X-ray examination of the os triquetrum will show no fracture in the antero-posterior view whereas the lateral and oblique views will; Bartone and Grieco[27] say much the same. Nevertheless Durbin,[29] in his demonstration of established non-union of a fracture of the triquetral bone, reproduces a radiograph showing clearly a transverse linear fracture of the bone at the initial examination in the antero-posterior view. This radiograph bears a close resemblance to the antero-posterior view taken of the carpal bones in my case where, also, the transverse linear fracture is readily seen. The lateral view is sometimes very informative in that it may show up a flake of bone that may vary in size from a minute fragment to one of 4 mm. in diameter;[25] it will also enable one to distinguish whether the fragment arises from the triquetral or the lunate.

A Case of Simultaneous Fractures of the Os Triquetrum and the Os Multangulum Majus in the Same Wrist

A youth of 16 years was travelling on a lorry which became involved in a road accident. He was thrown from the vehicle to the ground, which he struck with his right shoulder. When he was brought for examination it was easy to detect a fracture of the right clavicle, the fragments of which were grossly separated. Apart from some degree of shock, no other injury was seen. However, on the following day the lad

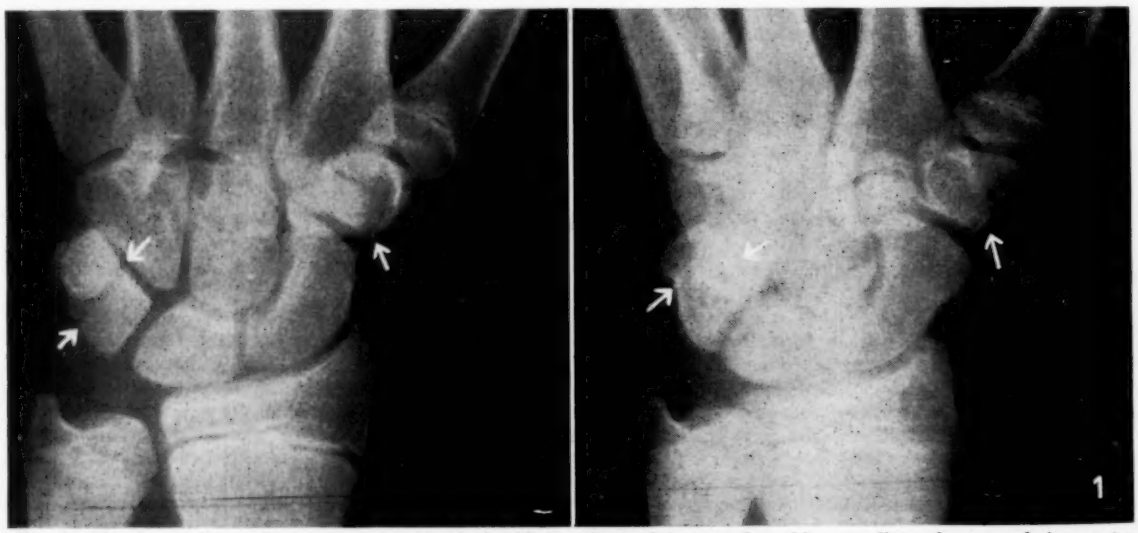

Fig. 1. Showing a linear fracture through the body of the triquetral bone and a chip or splinter fracture of the greater multangular bone in the antero-posterior and oblique views.

complained of some pain, swelling and stiffness in the left wrist-joint and an X-ray examination disclosed a transverse fracture through the body of the os triquetrum and a flake or splinter of bone avulsed from the os multangulum majus, these fractures showing up in both oblique and antero-posterior views (Fig. 1).

The Manner of Production of Fractures of the Os Triquetrum and Os Multangulum Majus

Greening[24] and Greening and Bonnin[28] go into the manner of production of fractures of the os triquetrum in detail. Their conclusions are summarized as follows: The fracture happens when a fall is taken upon the palm of the hand and a fairly large area of the dorsum of the triquetral is compressed between the pisiform bone and the lower end of the ulna, so producing a fracture extending through the body of the bone. But, if the triquetral bone is momentarily dislocated backwards, the ligament passing between it and the lunate avulses a flake of bone without a compression fracture. The os triquetrum is roughly pyramidal and its sharpest angle is inserted between the hamate and lunate bones. This wedged insertion tends to extrude the bone when falls are taken upon the extended wrist, but the tendency is counteracted by a movement of the wrist into ulnar deviation whereby the os triquetrum moves more deeply into the wrist under cover of the distal end of the ulna. Falls taken on the back of the hand, the wrist being violently flexed, impose an excessive strain on the ulnar collateral ligament and a flake is pulled off or the ligament is torn. It has been remarked upon that a fracture of the os triquetrum always accompanies a perilunate dislocation of the carpus, when all the bones of the wrist, except the lunate, are dislocated backwards and outwards so that a flake of bone is torn from the triquetral by ligaments which attach it to the lunate.

The os multangulum majus has been regarded as almost immune to fracture, and even experimental attempts to fracture the bone have not always been successful; yet it has been fractured, as the few reported cases show. Transverse fracture through the body have occurred but a flake type of fracture is commoner. The bone is so well protected by ligamentous attachments that fractures are rare, and it can be only a direct wrenching upon a ligament that avulses the bony fragment.

Symptoms

Even in the simplest types of fracture of the os triquetrum pain, tenderness, swelling and restriction of movement of the wrist occur. These symptoms may be slight or severe, and when they are slight they may give the clinical picture of a sprain of the wrist. Some authors have been able to localize symptoms over the bone itself but they are much more frequently spread over the region of the wrist, when it is reasonable to suspect some other injury than a simple fracture of the os triquetrum. In rare cases the fragments of bone have been actually palpated, but even where a fracture of the triquetral is strongly suspected it is not possible to differentiate the type of fracture. The evidence points to the necessity for radiographic differentiation of a sprain of the wrist from a fracture of the os triquetrum.

DISCUSSION

The literature indicates that fracture of the os triquetrum is uncommon and is rarely associated with fracture of another carpal bone. Simultaneous fracture of the os triquetrum and the os multangulum majus in the one wrist is most rare; I have not found a single case in a search through the relevant literature, and this case is presented as a unique instance. Had there been no accompanying fracture of the os multangulum majus it would still not be easy, despite Greening's explanation of the methods of production, to explain how a youth falling heavily upon his right shoulder and badly fracturing his right collar bone could also fracture the os triquetrum of his left wrist. It is even more difficult to explain the combined carpal fractures. However, disregarding the fracture of the clavicle, the tentative suggestion is that part of the impact of the fall was taken by the hand with the wrist in the fully extended position and in excessive ulnar deviation, which latter stress was taken up by the radial collateral ligament, which produced the flake type of fracture through its attachment to the greater multangular bone; and that the fracture through the triquetral bone was due to direct compression at the moment that the wrist was in this extreme position.

Although this case of triquetral fracture, like Durbin's, could be diagnosed on the antero-posterior radiographic examination, there is much evidence that all sprains of the wrist should, if possible, be X-rayed in the lateral and oblique positions as well as the antero-posterior. Failure to detect a fracture through the body of the triquetral by omission of these examinations leads to a painful non-union of the fragments, as Durbin[29] clearly showed. It appears likely that if all sprains of the wrist were X-rayed many more cases of fracture of carpal bones would be brought to light, and that those sprains of the wrist that take so long to heal and then leave the patient with a weakened wrist would be shown to have a bony pathology. There remains no doubt that whether there is or is not wide separation of the fragments, fixation of the wrist in the optimal position for function for at least 5 weeks is the right treatment.

SUMMARY

A review of the literature on the more unusual carpal fractures is presented as a background to a case of combined fractures of the triquetral and greater multangular bones in the one wrist. A survey of previously published classifications of triquetral bone injuries is made, significant points in radiography are mentioned, and a discussion is entered upon of the manner of production of these fractures, their symptomatology, and the respect with which all sprains of the wrist should be regarded.

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BRITISH COUNCIL COURSE : REHABILITATION OF THE LIMBLESS

The British Council are arranging for a course on 'Rehabilitation of the limbless' to be held at Queen Mary's Hospital, London, S.W. 15, from 12 to 25 June 1960.

The course will be directed by Dr. T. Ritchie, M.B., Ch.B. (Edin.) Principal Medical Officer, Ministry of Health, who is in charge of the medical work of the Ministry's limb-fitting service and he will be assisted by members of his staff. Most of the course will take place at Queen Mary's Hospital itself, but visits will be paid to factories making limbs and to the Camden Road Rehabilitation Centre, the National Spinal Injuries Centre, Stoke Mandveille, Buckinghamshire, and Queen Elizabeth's College, Leatherhead, Surrey.

The course is intended for surgeons and doctors engaged in work for amputees, and possibly for Government and other officials concerned with the rehabilitation of persons from such injuries.

There will be vacancies for 15 members only, and the cost, including accommodation, will be £47. A detailed prospectus will be available shortly from The Director, Courses Department, The British Council, 65 Davies Street, London, W. 1, to whom application for membership should be made.

SOUTH AFRICAN NATIONAL COUNCIL ON ALCOHOLISM

As part of a national drive to bring home to South Africa the devastation being caused in this country by alcoholism, SANCA is sponsoring a tour of the main centres of the Union by Dr. Marvin A. Block, B.S., M.D., during March 1960.

Dr. Block is a world authority on alcoholism, and Chairman of the American Medical Association's specially formed Committee on Alcoholism. He is also a Director of the National Council on Alcoholism, the American organization to which SANCA is affiliated. Dr. Block is also Assistant Clinical Professor of Medicine at the University of Buffalo Medical School, New York, and is a postgraduate of London, Paris, Berlin and Vienna. He is the author of a large number of articles, both scientific and popular, on problem drinking and is well known in the USA as a lecturer on this subject.

During his visit to South Africa, Dr. Block will give a series of public lectures, as well as addresses to specialized groups—medical practitioners, medical students and student nurses, social workers, industrialists, etc. His message will be that alcoholism is a treatable disease and not an antisocial complaint that must be kept hidden.

The following is a list of places at which Dr. Block will lecture after 15 March 1960. The programme of lectures at Johannesburg and Pretoria which took place before that date, was received too late for publication. It is suggested that interested doctors inquire at their local Branch or SANCA office for details of lectures not given here.

Friday 18 *March: Bloemfontein*
Evening: Public meeting.

Monday 19 *March—Friday* 26 *March: Cape Town and district*

Monday	12.00 p.m.	Physiology Lecture Theatre, Medical School, Observatory; for medical students
	8.15 p.m.	Public Meeting, National Hall, Fabriek Street, Paarl.
Tuesday	9.25 a.m.	Dutch Reformed Seminary, Stellenbosch; for theological students
	10.25 a.m.	University of Stellenbosch; for social science students
	3.30 p.m.	Murray Hall, Plein Street, Stellenbosch; for women's organizations.
	8.15 p.m.	Park Road Hospital, Rondebosch; College of General Practitioners.
Wednesday	12.00 p.m.	Karl Bremer Hospital, Bellville; for medical students
	8.15 p.m.	Physiology Lecture Theatre, Medical School, Observatory; Medical Association (Cape Western Branch)
Thursday	8.15 p.m.	Public meeting, Temple Israel Hall, Main Road, Green Point.
Friday	9.00 a.m.	Nursing College, Cape Town
	12.45 p.m.	Rotary Club, Cape Town.

Monday 28 *March: Port Elizabeth*
Afternoon: Public meeting.
Evening: Cape Midlands Branch, (M.A.S.A.)

Sunday 3 *April—Monday* 4 *April: Durban*

Sunday	8.30 p.m.	Public meeting.
Monday	12.45 p.m.	Association of Neurologists, Psychiatrists and Neurosurgeons of South Africa (M.A.S.A.).

Friday 22 *April: Salisbury, S.R.*
8.15 p.m. Mashonaland Branch, M.A.S.R, BMA.

Tuesday 26 *April: Bulawayo, S.R.*
Evening: Matabeleland Branch, M.A.S.R, BMA.

Saturday 30 *April: Kitwe, N.R.*
Evening: Congo Border Branch, BMA (provisional).

PASSING EVENTS : IN DIE VERBYGAAN

Pensions for Self-employed Persons. Members will have noted with interest the reference made by the Minister of Finance in his Budget Speech to the allowance of £300 *per annum*, which is available for pension purposes, to self-employed persons. Many pension schemes may be brought to the notice of practitioners in the immediate future and members are reminded that the Association is going into the question of a suitable pension scheme applicable to persons in private practice. In due course it is hoped that an announcement will be made in the *Journal*.

* * *

College of General Practitioners, Witwatersrand Faculty. The next meeting of this Faculty will take place in the Lecture Theatre of the South African Blood Transfusion House, Klein Street, Hospital Hill, Johannesburg, on Tuesday 29 March at 8 p.m. This will be the Annual General Meeting of the Faculty and will be followed by a small informal clinical meeting.

* * *

Dr. André Swanepoel, internis, het sy adres verander van National Mutual-Gebou na Mediese Sentrum 1002, Heerengracht, Kaapstad. Die telefoonnommer bly onveranderd: 3-4091.

Dr. André Swanepoel, physician, has changed his address from National Mutual Buildings to 1002 Medical Centre, Heerengracht, Cape Town. The telephone number remains unchanged: 3-4091.

* * *

Research Forum, University of Cape Town. A meeting of Research Forum will be held on Wednesday 16 March at 12 noon in the Bennie de Wet Lecture Theatre, A-floor, Groote Schuur Hospital, Observatory, Cape. Dr. S. Cohen, of Mill Hill, London, will speak on 'The metabolism of human gamma globulin'. All who are interested are invited to attend this meeting.

* * *

University of Cape Town and Association of Surgeons of South Africa (M.A.S.A.) Joint Lectures. The next lecture in this series will be held on Wednesday 16 March at 5.30 p.m. in the E-floor Lecture Theatre, Groote Schuur Hospital, Observatory, Cape. Dr. M. C. Botha will speak on 'Blood transfusion'. All members of the Medical Association are welcome.

* * *

South African Institute for Medical Research, Staff Scientific Meetings. A meeting will be held on Monday 14 March at 5.10 p.m. in the Institute Lecture Theatre. Dr. H. P. Weinbren will speak on 'The techniques used in the identification of arthropod-borne virus diseases'.

On Monday 21 March at 5.10 p.m. a meeting will be held in the Institute Lecture Theatre. Dr. H. D. Barnes will speak on 'Chromatography'.

IN MEMORIAM

THOMAS MULOCK-BENTLEY, L.R.C.P.I. & L.M., L.R.C.S.I. & L.M.

Dr. E. W. S. Deale, of Durban, writes:

It was my great privilege to know the late Dr. Mulock-Bentley for nearly 40 years. He was a man of sterling character, greatly loved by the many people into whose daily lives he entered as doctor and friend. He lived a full and happy life, and through the long years he practised as a doctor he was known for his knowledge and love of his fellow beings.

Dr. Mulock-Bentley

Born in 1866 in Dublin, he qualified at the Royal College of Physicians and Surgeons of Ireland in 1888. Two years later, for health reasons, he left England and joined the late Dr. Blood as a partner in Vrede in the Orange Free State.

At the outbreak of the Anglo-Boer War he was instructed by President Steyn to form an ambulance unit. At the battle of Spioenkop he attended the wounded from both the British and Boer armies with complete impartiality. When Bloemfontein capitulated he was given permission to join the RAMC and as a captain he served until peace was declared. For his services he was mentioned in dispatches and awarded the Queen's and King's medal. He resumed his local practice in Vrede until World War I when he served in the UDF, and yet again offered his services in World War II.

In 1893 Dr. Bentley married Norah Richter, who died in 1957. He had 3 sons who survive him. Notwithstanding his devotion to his practice, he was able to devote time to the civic affairs of his town, and was mayor for 2 years. He did not fail in his duty to his church and was churchwarden for many years. He was a keen Freemason, for many years attending his local lodge. Sport, too, claimed his attention; he played golf and bowls, and excellent tennis up to his 80th year.

He retired from practice in Vrede in 1925 leaving his practice to his eldest son, Dr. T. Mulock-Bentley Jr. His new home was in Durban and from here he went on several voyages as ship's surgeon. In Durban he made many new friends and held various appointments. He was a regular patron of horse racing and enjoyed this until a few days before his death.

It is amazing to realize that when he started practice, telephones and cars were unheard of and he carried on the practice in his large area by cart and horse, and often by horse only, carrying a good stock of medicines in a large wooden box. He astonished his patients by driving his first car in 1909, and there were many who felt that he was risking his life at the dangerous speed of 20 miles an hour.

Although he was denied the great developments in medicine that took place after he retired from practice, he truly upheld the honour and dignity of his profession. He was one of the early pioneers and a wonderful example of the grand and much loved family doctor.

THOMSON DUNCAN SHANKS, M.B., CH.B. (GLASG.)

Dr. T. D. Shanks died on 4 February 1960 after a long illness.

He qualified to the degree of M.B., Ch.B. at the University of Glasgow in 1924 and was in private practice in the UK until he came to South Africa in 1947. In Britain, Dr. Shanks held the position of Hon. Physician to the Gateshead Children's Hospital for 19 years, medical officer to the Briar Medical Children's Nursery for 18 months, medical officer to Gateshead South G.P.O. for 16 years, and Divisional Surgeon to the St. Johns Ambulance Brigade at Low Fell, Durham for 3 years, and at Gateshead for 15 years.

Dr. Shanks

On coming to South Africa Dr. Shanks entered general practice in Cape Town until 1955, where he was medical officer to the Mary Rolt Hostel, Mowbray, and also acted as a locum for the Health Department on one occasion.

Before taking up his position of Medical Officer at Sandflats Health Centre, where he died, Dr. Shanks was physician and surgeon to the Nessie Knight Hospital at Sulenkama in the Transkei.

Dr. P. C. Grenfell, District Surgeon, Alexandra, C.P., writes:

The passing of Dr. Thomson Duncan Shanks is being mourned by a very large circle of people of all races and creeds at Sandflats, C.P. During the 5 years that he served as Medical Officer of the Sandflats Health Centre his gift of understanding and sympathy for people in all walks of life endeared him to all who came in contact with him. He was in every sense of the word 'the beloved physician' whose wise counsel and quiet words of sympathy and encouragement brought comfort and encouragement to many.

I count it a privilege to have known a man of his stature, and Sandflats is the richer for those few years of his faithful service there.

My sympathy goes out to his widow and family.

NEW PREPARATIONS AND APPLIANCES : NUWE PREPARATE EN TOESTELLE

SURGICAL HAND LAMP

VEB Carl Zeiss Jena introduce their new Surgical Hand Lamp, and supply the following information:

The surgeon has at his disposal in the operating theatre large, almost shadowless operating lamps. For special operations additional lamps such as ball-reflectors and supplementary surgical illuminants are placed beyond the operating area and can be focused from there on the object.

For deep cavities, e.g. the thoracic cavity, or for illuminating the dome of the pleura, it is necessary to direct these lamps almost horizontally. It is therefore difficult and sometimes takes up much time to arrange these positions at the correct time. With the newly designed sterilizable surgical hand lamp the assisting surgeon can, without impeding the surgeon, illuminate these cavities. In cooperation with surgeons a surgical hand lamp was designed which can even be inserted into the thorax itself. All requirements regarding sterilizing capacity, low heat generation, intensity of illumination, illuminated area and serviceability could be satisfied. At the focus of a parabolic mirror there is a 5-watt 6-volt lamp mounted in a centring socket. The effective illuminated area with a diameter of 50 mm. has a central brightness of approximately 2,100 lux. The cover glass has a reflection-reducing coating. The removable sheath is chromium plated, the handle is made of black plastic. The lamp weighs approximately 170 gr. The flexible cord and the surgical hand lamp are covered with a sterile material cover.

It can be assumed that these lamps, after being positively tested and approved, will not only be required for surgery but will also be found useful by ear, nose and throat specialists and ophthalmic surgeons, etc.

Further information may be obtained from Messrs. Optolabor (Pty.) Ltd., P.O. Box 1820, Johannesburg, the South African agents for VEB Carl Zeiss Jena.

BOOK REVIEWS : BOEKBESPREKINGS

PATHOLOGY YEAR BOOK

The Year Book of Pathology and Clinical Pathology 1958-59. Edited by W. B. Wartman, B.S., M.C. Pp. 502. 138 figures. $8.50. Chicago: The Year Book Publishers, Inc. 1959.

In the inimitable manner that has characterized the previous Year Books, the editor, Prof. Wartman, takes us through a series of articles in pathology and clinical pathology which were published mainly in the medical press of North America and Europe. They are generally grouped as touching upon allied classes of subjects.

The cardiovascular system group has a provocative account of the cardiac necrosis that occurs in rats under corticoid therapy when treated with sodium phosphate, and also good accounts (with footnotes) on the natural history of atherosclerosis, the mast cells in coronary atheroma, and regenerative changes in the aorta and occluded vessels.

Under the respiratory system are rightly emphasized the emphysematous and pulmonary hypertensive disturbance, and some possibly allergic connective-tissue diseases of the lungs.

The Nuffield Trust investigation into sterilization is extracted to highlight the flaws in medical cleanliness—where for example 36 out of 209 allegedly sterile articles used in wards and theatres were unsterile; and the pollution of bedside carafes is exposed. However, the sections of forensic pathology, urinalysis and virology are poor, and only the editor's footnotes saves the section on leucocytes—where the plea for simpler preliminary investigations in clinical pathology is a pertinent one.

Reading this Year Book, the importance of immunity in the basic understanding of medicine again appears as a distinct trend in pathology and clinical pathology.

The book maintains its excellent value to the academic pathologist. While clinical pathologists may miss some reviewing of practical matters, for them also the Year Book continues to be an invaluable and recommended acquisition. R.S.

ENDOCRINOLOGY IN GYNAECOLOGY

Gynecologic Endocrinology. By G. N. Riley, Ph.D. Pp. xix + 330. 72 figures. $8.50. New York: Paul B. Hoeber, Inc. 1959.

The struggle to avoid the quicksands of empirical endocrinal treatment and stand unshakably firm on physiopathological knowledge in the approach to disorders of menstruation, the menopause, and infertility, is all too seldom made and sustained. To those whose spirit remains willing but whose flesh is too weak to cope with the bewildering literature and rival claims of proprietary preparations in this field, this book will provide a welcome modern aid to informed and rational treatment. It is even sufficiently comprehensive to be of value to those with aspirations towards diplomas in gynaecology and obstetrics.

The clarity of the type, quality of the paper, and binding of this volume are a credit to the publishers.

The author, who is an Associate Professor of Gynecology and Obstetrics of the University of Michigan, has a lucid literary style, and the excellence and clarity of no less than 72 figures help to hold the reader's attention and enhance the concise presentation of the subject material.

The opening chapters deal with endocrinal and reproductive physiology in women. The physiopathology and rational treatment of menstrual dysfunction, the endocrine aspects of infertility, abortion and adrenal cortical function, *inter alia*, are valuable practical guides. Notwithstanding that some of the details provided in the ensuing chapter on diagnostic procedures are beyond the requirements of practitioners, they are easily understood, and the closing chapter, which provides details on pharmaceutical endocrine preparations, can be recommended as particularly helpful. D.C.

CHILD PSYCHIATRY

An Introduction to Child Psychiatry. By S. Chess, M.D. Pp. x + 254. $5.25. New York and London: Grune & Stratton, Inc. 1959.

Child psychiatry is a field where many disciplines meet—health, education and the social sciences. Because of the simplicity of its language and its middle-of-the-way approach, this excellent little book can well serve as an introduction to anybody interested in this comparatively new field.

It is concise and touches on the most important aspects of child psychiatry. The only criticism that can be levelled at it is that the child is considered too much in isolation from the parent, and the dynamic interaction between them is largely overlooked. This gives a deceptive impression of simplicity, as though counselling is all the parents require.

In the section on classification and diagnosis Dr. Chess took considerable pains to clarify some difficult and confused concepts. Each chapter is followed by a useful bibliography, which the interested student can explore. As an introduction it can be recommended without reservations. M.V.B.

PLASTIC SURGERY OF THE FACE

The Surgical Treatment of Facial Injuries. 2nd edition. By V. H. Kazanjian, *C.M.G.*, D.M.D., M.D., F.A.C.S., D.Sc. (Hon.) and J. M. Converse, M.D., F.A.C.S. Pp. ix + 1110. 1,155 figures. 176s. $22.00. London: Baillière, Tindall and Cox Ltd. Baltimore: The Williams & Wilkins Company. *1959.*

Dr. Kazanjian is one of the diminishing band of surgeons whose work extends back to World War I, when he worked at Sydenham with Sir Harold Gillies, and his experience of facial injuries must be almost unique. His co-author, Dr. Converse, also worked with Sir Harold, in World War II.

The book forms a most comprehensive study of facial injuries of all types. The authors stress the importance of early correction of facial fractures for the reason that they set rapidly and after a few days are most difficult to correct. Unfortunately, in our country, with its long distances, it is not uncommon to find that these fractures have united firmly in a bad position by the time they reach a plastic centre.

The authors also stress their preference for repair by local flaps, where possible. These permit of shorter treatment with far better colour match.

The book is excellently illustrated with photographs and diagrams of the various procedures and is a great contribution to plastic surgery. It should be on the shelf of every surgeon interested in this work. N.A.M.P.

POISONS AND POISONING

Klinik und Therapie der Vergiftungen. 3., neubearbeitete und erweiterte Auflage. Von Priv.-Doz. Dr. S. Moeschlin. Pp. xii + 655. 92 Abbildungen. Ganzleinen DM 59.00. Stuttgart: Georg Thieme Verlag. 1959.

The entire volume is concerned with drugs and poisons and clinical manifestations in cases of overdosage. It also deals with numerous very important industrial hazards to which the worker is exposed in modern industry. Special emphasis is laid on dangers from fruit sprays and modern insecticides. Unlike most text-books of pharmacology, this publication deals also with treatment and prevention.

An excellent résumé is included on lead poisoning and new drugs employed in its treatment. Calcium EDTA is labelled as specific against lead poisoning in addition to BAL.

In conclusion, one cannot help emphasizing the significance of prevention in industrial hazards, as pointed out by the author, who converts this nebulous subject into one of pleasant reading and easy understanding. D.J.H.

GASTRO-ENTEROLOGY

Proceedings of the World Congress of Gastroenterology and the 59th Annual Meeting of the American Gastroenterological Association, Washington, D.C., USA, 25 - 31 May 1958. Vols. 1 and 2. Pp. xxxv + 1,363. Illustrated. 160s. London: Baillière, Tindall and Cox Ltd. 1959.

These two volumes contain original contributions, papers read in symposia, and reports of panel discussions. In addition there is a description of scientific exhibits, a list of films shown at the Congress, and an account of the inaugural ceremony.

As a source of reference to original contributions the value and necessity of this record is evident; but the symposia and panel discussions provide more than this, for they form a combined account of various aspects of gastro-enterology from authorities from all over the world. This is well illustrated by the symposium on peptic ulcer, where aetiological aspects of the disease can be compared against the geographical, social and dietary backgrounds of a large number of different countries.

The book is attractively prepared with clear print on good paper and the reproduction of the illustrations is excellent. A.J.W.

MEDICAL BIOGRAPHY

Notable Names in Medicine and Surgery. 3rd edition. By Hamilton Bailey, F.R.C.S. (Eng.), F.A.C.S., F.R.S. (Edin.), and W. J. Bishop, F.L.A. Pp. xiii + 216. 79 portraits and 162 other illustrations. Price in Great Britain: 35s. London: H. K. Lewis & Co. Ltd. 1959.

Many practitioners and students have wished that they knew more of the persons whose names they use almost daily in their work or reading. The third edition of this work has been greatly expanded and includes many more names than the previous edition. Where there were formerly more notes on persons connected with surgery than with medicine, this has now been remedied. Footnotes concerning persons mentioned in the text are included and the whole makes interesting reading, containing as it does fascinating glimpses of little-known facts of medical history.

A.H.T.

PANCREATIC TUMOURS

Tumors of the Pancreas. By V. K. Frantz, M.D. Pp. 149. Illustrations. $1.50. Washington: Armed Forces Institute of Pathology. 1959.

The *Atlas of Tumour Pathology* produced by the Armed Forces Institute of Pathology, Washington, D.C., is well known to most pathologists. The latest fascicle *Tumours of the Pancreas* exhibits the same care in preparation and excellence of the illustrations that characterized its predecessors. The author brings us completely up to date with the many problems of pancreatic neoplasia. She exhibits a healthy scepticism of many so-called facts which on proper enquiry are found to be time-encrusted theories.

The clinical interest attached to many of the pancreatic neoplasms, particularly the functioning types, is so great that the reviewer can thoroughly recommend the fascicle to all. Not the least attractive feature is the comprehensive list of references attached to each section.

B.B.

CANCER OF THE SKIN

Cancer of the Skin. By John C. Belisario, *C.B.E.*, E.D., M.D., Ch.M., D.D.M. Pp. xvii + 321 + (14). 201 figures. 57s. 6d. + 1s. postage. London: Butterworth & Co. (Publishers) Ltd. South African Office: Butterworth & Co. (Africa) Ltd., P.O. Box 792, Durban. 1959.

It cannot be too strongly stressed that this book should be widely read and carefully studied in this country. Dr. Belisario proceeds to describe in detail and with magnificent clarity the various skin cancers that are so commonly encountered, both in Australia, where he practises, and in South Africa.

The photographs are extremely well done and the reproduction leaves nothing to be desired. It is a pleasure to see and to read the book, which does great credit to the publishers as well as to the author.

Everybody who deals with many cases of skin cancer develops methods of treatment to suit himself and, by a process of trial and error, satisfies himself that his method is the best with the facilities available. The reviewer cannot agree that scraping rodent ulcers should play a serious part in this therapy, and strongly supports the monobloc operation devised by Pack for malignant melanoma.

But these are small criticisms. The book is a landmark and a vital contribution to a very important subject.

T.S.

STRABISMUS

Squint and Allied Conditions. By George P. Guibor, M.D., D.D.S. Pp. xi + 356. 103 figures. $11.50. New York and London: Grune & Stratton, Inc. 1959.

The author, in a study of over 41,000 patients, claims a remarkable percentage of cures in cases of squint with his routine of non-surgical treatment. The basic points are the use of atropine and prisms and judicious occlusion and orthoptics. Unfortunately he gives very few indications of his criteria of cure apart from parallelism of the visual axes. This system of therapy must therefore be considered as unorthodox and highly individual. His results appear certainly no worse than the usual results obtained and in many cases they seem superior. His methods are therefore worth a trial, especially as considerably less surgery is done. Unfortunately our patients seem less cooperative than his and parents are unwilling to wait long months or years for results.

Certain of his statements go completely counter to orthodox teaching, such as tackling as many as 8 muscles at one operative session, or occlusion for one or more hours a day. Others will be subscribed to by few. Thus, he rarely operates before the age of 8 and rarely finds it necessary to operate more than once to correct a squint. Again, bilateral medial-rectus retroplacement is reserved for bilateral abducens palsy, and his usual operative procedure for a concomitant convergent strabismus consists of 1 mm. retroplacement of medial rectus and bilateral external-rectus resection of 10 mm. Most operations are done with local anaesthesia. There are, however, many useful observations, such as his stress on the frequency of supranuclear lesions and the value of atropine and prisms.

In short, this is a highly controversial book in which a great plea is made for conservatism in treatment, mainly with atropine and prisms. The claims made should not be lightly dismissed, but warrant further investigation.

L.S.

BACTERIOLOGY: ITS BIOLOGICAL AND CHEMICAL BACKGROUND

Basic Bacteriology. Its biological and chemical background. 2nd edition. By Carl Lamanna, Ph.D. and M. Frank Mallette, Ph.D. Pp. xiii + 853. Illustrations. 108s. and $13.50. London: Baillière, Tindall and Cox Ltd. Baltimore: The Williams and Wilkins Company. 1959.

The second edition of this book is now available and certain sections have been thoroughly revised and brought up to date. It deals with some fundamental biological and chemical aspects of bacteria. The section on bacterial metabolism, which constitutes the largest single part of the text, is particularly well covered, and there is also a useful chapter on bacterial genetics and variation. The section on chemical disinfection could perhaps have been expanded to give more detailed consideration of antibiotics.

The book is intended as a work of complexity intermediate between the elementary texts and the erudite monograph and in this respect it fulfils its function adequately. The subject matter, however, is too detailed for the average undergraduate student and somewhat belies the title of *Basic Bacteriology*.

K.C.W.

MEDICAL TREATMENT OF DISEASES OF THE THYROID

Die Medikamentose Behandlung der Schilddrüsenerkrankungen. Von Prof. Dr. W. Grab und Prof. Dr. K. Oberdisse. Pp. xii + 285. 55 Abbildungen. 14 Tabellen. DM 39.60. Stuttgart: Georg Thieme Verlag. 1959.

Diseases of the thyroid are the commonest endocrine diseases of today, and are more frequently diagnosed because of improved diagnostic methods. These cases are commonly seen in practice by physicians, and this book was written with that in view. The first half deals with normal and pathological physiology of the thyroid and with drugs that influence thyroid function. Grab, who writes this section, explains the morphology and function of the thyroid and its influence on other organs and on general metabolism. The second part of this concise and clear treatise is devoted to the action of drugs used in diseases of the thyroid. In this section Oberdisse discusses diagnostic methods and the treatment of hyper- and hypothyroidism. He mentions thiouracil and its derivatives and suggests their selection in particular cases. The treatment with radio-active iodine is explained, and so is the prophylactic treatment with iodine in cases of endemic goitre etc. The book is written in clear and easy German and should be of interest, especially to general practitioners and physicians.

H.W.W.

OBSTETRIC ANAESTHESIA

Principles and Practice of Obstetric Anaesthesia. By J. Selwyn Crawford, M.B., Ch.B., D.A. (England), F.F.A.R.C.S. Pp. 128. Illustrations. 20s. Oxford: Blackwell Scientific Publications. 1959.

Being the first British text-book of obstetric anaesthesia that has been published for some time, this excellently-produced concise little book reflects the trends and evaluates the methods of obstetric analgesia and anaesthesia now practised in Great

Britain. There is also an interesting chapter on the physiological changes accompanying pregnancy, such as those involving the blood and body fluids, the heart and vessels, respiration, digestion, the renal tract, and biochemistry.

In the section dealing with the placenta, the author reviews the functions of this most mystifying of all the organs in the body and goes with great detail into the transmission of the respiratory gases, with special reference to the average oxygen content of the uterine and umbilical vessels, as well as the haemodynamics. The placental transmission of the various anaesthetic agents is also discussed at great length and, though of academic importance, will not greatly interest the general practitioner.

The author comprehensively reviews the various analgesic and anaesthetic methods in use and devotes a separate chapter to anaesthesia in obstetrical complications. The physiological aspects of perinatal life are discussed, especially as they influence the work of the obstetric anaesthetist.

Altogether this is a most interesting, readable and instructive book, which can be highly recommended for all practitioners.

A.M.M.

SOCIAL PSYCHOTHERAPY

Progress in Psychotherapy. Vol. 4. *Social Psychotherapy.* Edited by Jules H. Masserman, M.D. and J. L. Moreno, M.D. Pp. xi + 361. Illustrations. $8.75. New York and London: Grune & Stratton, Inc. 1959.

The flood of medical literature calls for periodical surveys, most of which are unsatisfactory in that they are scrappy and attempt to cover too much ground. Masserman and Moreno have found a solution to this problem by publishing short articles on fairly circumscribed subjects each year. The present volume, with over 40 articles, is a little uneven but it is very well worth reading. An article on economics and psychiatry is somewhat unflattering in its views on Soviet psychiatry but gives some interesting glimpses concerning television and other technicological advances on the psychiatric patterns of the community. Torrence's article on mutual support and sacrifice in groups under stress and Opler's views on anthropology and psychiatry are short but lively pointers to further reading. Moreno's verbatim report on the psychodrama of a dream makes fascinating reading and suggests a technique which might well be helpful with many patients. Ronald Albino, of the University of Natal, has written on some of the problems of psychopathology which face anyone who wishes to study group and racial differences in South Africa, and although he rightly avoids sweeping generalizations he provides a useful list of references and stresses the importance of nutritional and educational differences rather than possible genetic factors.

The whole volume is well up to the standard of its predecessors and a useful addition to the library of anyone interested in the widening frontiers of psychiatry.

J.M.MacG.

LOW BACK PAIN

The Degenerative Back and its Differential Diagnosis. By P. R. J. M. Hanraets, M.D. Pp. xi + 690. 315 illustrations (12 in colour). 53 tables. 105s. Amsterdam, London, New York, Princeton: Elsevier Publishing Company. 1959.

This volume elaborates in the greatest detail the theme that some individuals are liable to low back pain. With infinite care, patience and often repetition the author analyses the reasons for this. Constitution, anatomy, pathology are surveyed and the differential diagnosis is discussed with frightening complexity.

The author has performed 2,000 laminectomies for low back disorders in about 10 years, and this has enabled him to describe many anomalies, which are beautifully illustrated with coloured plates.

It is a pity that many errors in translation are apparent throughout the book. The printing, line-drawings and the radiographs are excellent.

A comprehensive bibliography and subject index is appended at the end of the book.

Dr. Hanraets has made a great contribution to the further understanding of a very common and difficult problem. M.S.

CORRESPONDENCE : BRIEWERUBRIEK

ABOLITION OF THE PREFERENTIAL MEDICAL AID SOCIETY TARIFF

To the Editor: The letter of Dr. Martyn[1] cannot be allowed to pass without comment, because it reflects views which are so often expressed by members of the Association, who are not fully aware of the background to the medical aid movement, or who were not in practice in the days when medical aid societies were few in number. Firstly it should be stated that medical aid societies were not created to 'give assistance only to those of the population who *cannot* (my italics) afford medical treatment'. They were established to assist persons who would find it difficult to meet their commitments, hence the term medical 'aid'. In other words, members of societies have to contribute a varying proportion of the accounts for medical services paid on their behalf by their societies.

Once more the hackneyed phrase is used of the doctor subsidizing industry, which is likely to influence the uninitiated to agree with Dr. Martyn. I wonder if members of the Association are aware of the fact that 75% of the members of medical aid societies earn a basic salary of £600 and less *per annum*, or that in the case of a certain society 75% of the members earn £35 or less per month. How can anyone in that income group pay for medical services unless his subscription to a medical aid society was brought within his means by the subsidy provided by the employer? Is the doctor subsidizing the employer or is the employer subsidizing the employee so that he may be able to pay the doctor, through the society, a reasonable fee for services rendered? If a few persons earning high incomes are included in the societies, their number is so small as to be negligible, compared to the large number of people who are able to pay for medical services instead of being hospital cases.

Dr. Martyn is not the first to advocate the abolition of the tariff and not likely to be the last, but it should be pointed out that the tariff was originally brought into existence not so much by the desire of the medical aid societies as by the pressure exerted by the rank and file of the profession. I have not the slightest doubt that if the tariff were abolished, not 6 months would elapse before the members of the Association would be clamouring for a return to the present system. It was the individual practitioners who, unbeknown to one another, in the first instance asked medical aid societies to pay them direct and in full in return for a discount. The request was acceded to, and that eventually lead up to the establishment of the tariff. There is no reason to assume that history will not repeat itself in this connection, which it is already doing as will be seen further on.

Certain medical insurance schemes have come into the picture lately and the Association is examining the position very carefully. The insurance schemes have been trying to obtain a tariff on which to base their actuarial calculations and have quite naturally approached doctors for the benefit of the preferential tariff, because their members, for the most, part fall within the same income category as members of approved medical aid societies. Furthermore, *doctors are once again pressing for payment direct to themselves* instead of to the member (exactly as was done in the earliest days of medical aid societies) and have given the insurance schemes to understand that they would accept fees according to the tariff. By acceding to this request the insurance schemes increase their costs of administration and consequently are seeking for a preference in fees.

The Central Committee for Contract Practice consisting of people knowledgeable in medical aid society affairs who are fully aware of all the issues, should be trusted to give the Association the benefit of their knowledge. The Federal Council consisting of the chosen delegates from the various Branches, may also be safely entrusted with the affairs of the Association and to decide what is best for the public as well as for the profession.

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23 February 1960

1. Correspondence (1960): S. Afr. Med. J., 34, 148.